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AN ANALYSIS OF ENVIRONMENTAL AND OTHER FACTORS ASSOCIATED WITH DEATHS FROM GASTRO-ENTERITIS IN CAPE TOWN IN 1956

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Families where deaths of young children have occurred are visited by the health visitors so that advice on hygiene and feeding may be given in respect of the other children. In order to obtain information about possible aetiological factors related to gastro-enteritis, enquiry forms were filled in by the health visitors in 250 consecutive deaths of children certified as due to gastro-enteritis in 1956. Most of the information given below is taken from these enquiry forms; it was collected in the following manner:

1. Observation of the environment by the health visitor.
2. History as given by the mother or other relative.
3. Information taken from death certificate, and from facts known to the medical officers and health visitors from previous observation of the children who had died.

The infantile mortality rate per 1,000 live births for the municipal area of Cape Town for 1956 was: from all causes, Europeans 24·6, non-Europeans 102·9; from gastro-enteritis alone, Europeans 3·1, non-Europeans 42·2.[1]

The deaths during the year show a marked seasonal incidence, being highest in the late summer months, February and March, and lowest in September. This is shown by the chart in Fig. 1.[1] It is not possible to give similar information for morbidity because cases of gastro-enteritis are not notifiable, but it is assumed that it is parallel with the mortality. The seasonal variation points to the importance of infection, for the disease is at its height when the weather is dry, dusty and hot, and flies are plentiful.

The home environment in the 250 deaths analysed is shown in Table I. The high proportion of deaths occurring where there is overcrowding, poor water-supply and sanitation, and a dirty neighbourhood predisposing to fly-breeding, again point to the part infection plays in the causation of the disease. The mother was working or ill in hospital in 58 cases (23·2%), the children being in the care of relatives or foster-mothers. The remainder were all cared for by their own mothers. Fifty children (20%) were reported to have been breast-fed. From observation at the child welfare clinics many of the breast-fed infants who contract gastro-

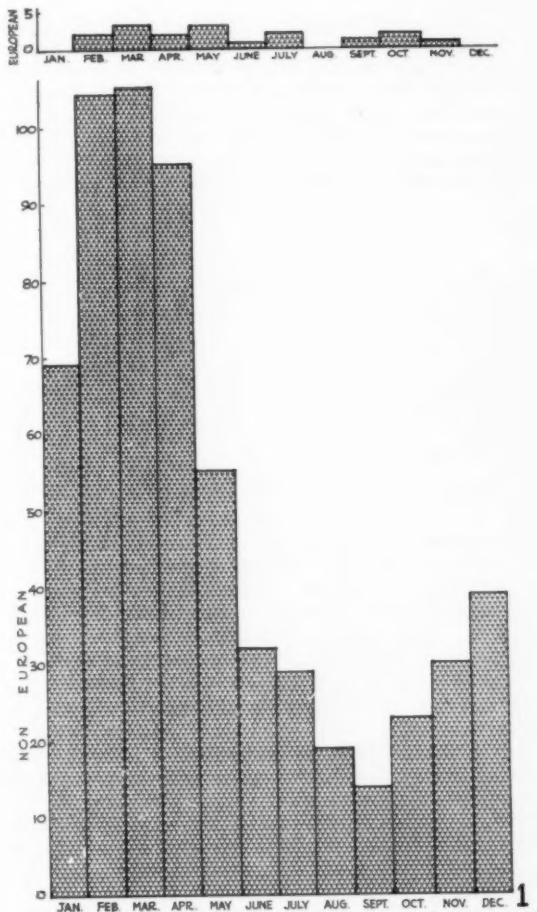

Fig. 1. City of Cape Town 1956. Seasonal incidence of deaths from gastro-enteritis.

* Arranged from information collected by the medical officers and health visitors of the Child Welfare Branch of the City Health Department, Cape Town.

enteritis are, from an early age, given unhygienic bottles of water, sugar-water or strained oats as well as the breast milk.

The children had been regular attendants at the child welfare clinics in 58 cases, (23·2%), attended occasionally

TABLE I

	No. of Cases	Percentage
Housing		
Brick house, no overcrowding ..	24	9·6
Brick house, overcrowding	105	42·0
Shed or tin shack	113	45·2
Sanitation		
Water borne	117	46·8
Pail closet	80	32·0
Nil	42	16·8
Water supply		
Taps in house	80	32·0
Standpipe near dwelling	81	32·4
Water purchased in tins	79	31·6
Neighbourhood		
Clean	72	28·8
Garbage lying about	138	55·2
No made roads	49	19·6

in 75 cases (30%), and never attended in 136 cases (54·4%). When it is realized that 92% of all non-European infants born in Cape Town attend child welfare centres,[1] the significance of these figures becomes obvious.

The contributory causes of death, as shown on the death certificates and from what is known of the cases, are shown in Table II. The chief fact which emerges from these figures

TABLE II

Contributory Causes of Death	*Certified*	*Known History*	*Total*	%
Acidosis, Shock, Dehydration ..	6	—	6	2·2
Kwashiorkor	4	2	6	2·2
Chronic or Recurrent gastro-enteritis	10	84	94	37·6
Malnutrition	17	47	64	25·6
Bronchopneumonia	24	3	27	10·8
Other Infections	12	—	12	4·4
Prematurity	7	3	10	4·0
Congenital Defects	8	—	8	3·2
Rickets	5	—	5	2·0
Roundworms	1	—	1	0·4

is the number of children who had chronic or relapsing diarrhoea for some weeks before death, and the number who were under-nourished. These figures are probably lower than the truth, as there was very little known of the history in many instances, and mothers commonly do not recognize under-nourishment in their own children.

Children dying in the nursing homes in which they were born, having never been home, numbered 6. Of these, 3 were premature.

Four Children died in the Place of Safety. These children were presumably all mal-nourished on admission, as children in need of care frequently are.

The age incidence is shown in the chart in Fig. 2, the maximum number of deaths occurring between 3 and 4 months, an age at which bottle feeding is commenced in many infants.

SUMMARY

The aetiological factors associated with gastro-enteritis were studied in 250 children who died of this disease in Cape Town in 1956.

Fig. 2. City of Cape Town 1956. Age incidence of deaths from gastro-enteritis in 250 cases studied.

There was found to be a seasonal incidence, the peak occurring in February and March.

Most of the homes in the cases studied were found to be overcrowded and the water-supply and sanitation were poor in the majority of cases. Although 92% of non-European children in Cape Town attend the child welfare centres, more than half the children who died had never attended any clinic. The contributory causes of death showed that a high percentage of the children who died had suffered from malnutrition and/or chronic or relapsing diarrhoea for some weeks before death. The age incidence showed that most deaths occurred round about 4 months of age. The facts studied illustrate the importance both of infection associated with unhygienic home conditions, and of susceptibility to the disease caused by malnutrition.

We wish to express our thanks to Dr. E. D. Cooper, Medical Officer of Health, Cape Town, for permission to publish the information contained in this paper.

REFERENCE

1. Annual Report of Medical Officer of Health, Cape Town for 1956 (in press).

Suid-Afrikaanse Tydskrif vir Geneeskunde

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VAN DIE REDAKSIE

EERSTE EKSAMENS VAN DIE KOLLEGE VAN INTERNISTE EN CHIRURGE VAN SUID-AFRIKA

Die Kollege van Interniste en Chirurge van Suid-Afrika het aangekondig dat hulle van voorneme is om hulle eerste na-graadse eksamens in Oktober 1957 te hou. Die eksamens wat suksesvolle kandidate toegang tot die Kollege sal verleen, is die Primêre eksamen vir die Fellowship van die Kollege van Chirurge (Suid-Afrika), die Fellowship van die Kollege van Interniste (Suid-Afrika) en die Fellowship van die Kollege van Verloskundiges en Ginekoloë (Suid-Afrika).

Op sy vergadering in Maart 1957 het die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad dit in beginsel goedgekeur dat hierdie drie hoër kwalifikasies as ‚addisionele kwalifikasies' erken word. Toe hierdie saak aan die Suid-Afrikaanse Geneeskundige Raad voorgelê is, was die presiese benamings van al die kwalifikasies nog nie vasgestel nie, en daarom was dit nie moontlik om 'n formele aanbeveling tot die Minister van Gesondheid te rig om die hoër kwalifikasies as addisionele kwalifikasies in die Staatskoerant aan te kondig nie. Die benamings, soos êrens anders in hierdie uitgawe aangekondig word (bl. xxv), is nou deur die Raad van die Kollege ooreengekom.

Dit moet duidelik verstaan word dat die kwalifikasies van die Kollege as *‚addisionele'* kwalifikasies in beginsel aangeneem is. Die kwessie van hulle aanbeveling deur die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad vir aanname en proklamering deur die Minister as *hoër* kwalifikasies vir toelatingsdoeleindes tot die statutêre Register van Spesialiste van die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad, is nog onder bespreking deur die betrokke komitees van die Geneeskundige Raad.

Die eerste Primêre eksamen vir die F.C.S. (S.A.)* sal deur sir Harry Platt, President van die Royal College of Surgeons of England bygewoon word, en die hoop word gekoester dat die eerste eksamen vir die Fellowship vir die Kollege van Interniste (S.A.) deur sir Russell Brain, President van the Royal College of Physicians of London, bygewoon sal word. Die eksaminatore sal gekies word uit 'n naamlys wat alreeds deur die Raad van die Kollege opgestel is en sal eksaminatore insluit met ondervinding van beide die standaard van hoër kwalifikasies van Suid-Afrikaanse Universiteite wat alreeds as hoër kwalifikasies vir die doeleindes van die Register van Spesialiste erken word, en die standaarde van oorsese eksamens van gelyke gehalte.

Dit is moeilik om eksamenstandaarde vas te stel, anders dan deur hierdie eksamens te hou wat toelaat dat die standaard bestudeer kan word. Die standaard van die Primêre F.C.S. (S.A.) sal sodanig wees dat dit deur die Royal College of Surgeons of Edinburgh en dié van Engeland aanneemlik sal wees vir vrystelling van die Primêre eksamens vir die Fellowships van hierdie twee Royal Kolleges. Enige twyfel wat toekomstige kandidate vir die Primêre F.C.S. (S.A.) mag hê aangaande die uiteindelike aanname van die Finale F.C.S. (S.A.) deur die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad as 'n hoër kwalifikasie in terme van die Register van Spesialiste, sal verminder word deur die wete dat hulle, nadat hulle die Primêre F.C.S. (S.A.) afgelê het, ten minste in staat sal wees om oorsee te gaan en vir die Finale Eksamens van die Royal College of Surgeons of England of dié van Edinburgh in te skryf, beide waarvan as hoër kwalifikasies volgens die Suid-Afrikaanse spesialiste-reglemente registreerbaar is.

Daar is geen primêre eksamen vir die F.C.P. (S.A.) en die F.C.O.G. (S.A.) nie. Die Raad van die Kollege het nogtans, deur aan te kondig dat hulle hierdie twee eksamens gaan hou, toekomstige kandidate die geleentheid gegee om vir die eksamens in te skryf, met die vertroue dat hierdie kwalifikasies in die toekoms by die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad as addisionele kwalifikasies en, na verwagting, ook as hoër kwalifikasies, registreerbaar sal wees.

Die eerste eksamens sal in Kaapstad gehou word. Toekomstige Kollege-eksamens sal in verskillende sentrums in Suid-Afrika gehou word. Die hoofrede waarom Kaapstad vir die eerste eksamens gekies is, is omdat die Universiteit, Kaapstad instruksie-kursusse in Anatomie, Fisiologie en Patologie in voorbereiding vir die eksamen aan kandidate vir die Primêre F.C.S. (S.A.) aangebied het.

Alhoewel dit nie streng van toepassing is op die onderwerp van sy eksamens nie, het die Kollege-Raad aangekondig dat die Registrateur van Maatskappye dit goedgekeur het dat daar 'n afdeling van Verloskunde en Ginekologie by die Kollege van Interniste en Chirurge van Suid-Afrika ingesluit word, gelyk in status aan die afdelings van Chirurgie en Interne Geneeskunde. Origens sal die Raad van die Kollege aanbevelings by die tweede Jaarlikse Algemene Vergadering van die Kollege in September 1957 voorlê aangaande die benaming van die Kollege, met die doel om nie alleen ‚Interniste en Chirurge' daarby in te sluit

*Die Engelse voorletters word dwarsdeur hierdie artikel gebruik.

nie, maar ook ‚Verloskundiges en/of Ginekoloë' of, andersins, om 'n meer omvattende en korter benaming te hê wat die drie hooftakke van Interne Geneeskunde, Chirurgie en Verloskunde en Ginekologie sal dek, sonder om een daarvan spesifiek te noem.

Die aankondiging van die Kollege-eksamens is 'n uiting van die Kollege se vertroue in sy eie doelwitte, en die ondersteuning wat die eksamens in die toekoms sal geniet sal 'n aanduiding wees dat een van sy belangrikste oogmerke verwesenlik is.

EDITORIAL

SARCOIDOSIS: CALCIUM AND THE KIDNEYS

Sarcoidosis is not a rare disease, and a suspicion exists that in South Africa it is commoner than elsewhere. Unfortunately, many sufferers from it languish for months or years with their condition undiagnosed—often in a sanatorium for tuberculosis. The usual manifestations include cutaneous and pulmonary lesions, lymphadenopathy, especially at the lung hila, iritis which may be part of a 'uveo-parotitis', hepatomegaly, and splenomegaly. Diagnostic measures include biopsy of skin, lymph nodes or liver. Raised serum globulin is common. X-rays of the hands occasionally show evidence of sarcoid infiltration. The well-known and more common appearance is that of multiple round 'punched-out' areas in the small bones; sometimes some of the phalanges appear thickened, coarsely trabeculated, and demineralized. Although frequently looked for, osseous lesions are distinctly uncommon.

A diagnostic procedure of which the value is not sufficiently appreciated is the simple estimation of the calcium in the serum or urine. Abnormally high calcium values are found far more often than the skeletal lesions mentioned above. The least that should be done is the simple Sulkowitch test on an early-morning sample of urine. This test has been discussed in an editorial in this *Journal*,[1] and is performed by mixing equal volumes of urine and Sulkowitch reagent in a test tube and allowing to stand for 3 minutes. If at the end of that time a thick cloud (of calcium oxalate) appears in the liquid then there is probably an excessive urinary excretion of calcium. The abnormality in calcium metabolism may then be confirmed by the estimation of the serum level and of the calcium content of a 24-hour collection of urine.

Consequent upon prolonged hypercalcaemia and hypercalciuria, calcium may be deposited in the renal parenchyma (nephrocalcinosis) and renal calculi may be formed. Severe renal damage is produced and death from uraemia is then imminent. The other great danger is to the eyes. Calcific deposition may occur in the cornea ('band keratopathy'), conjunctiva and sclera, with consequent loss of sight.

Davidson *et al.*[2] report further sequalae to the hypercalcaemic state, namely peri-articular soft-tissue calcifications around the shoulder and hip joints and premature arterial calcification in young people. Scholz and Keating,[3] from the Mayo Clinic, have recently discussed the renal disorder in sarcoidosis. Both groups of authors point out that, although granulomatous sarcoid deposits may actually occur in

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SARKOÏDOSE; KALSIUM EN DIE NIERE

Sarkoïdose is nie 'n seldsame siekte nie en daar is 'n vermoede dat dit baie meer algemeen in Suid-Afrika as êrens anders voorkom. Ongelukkig kwyn baie lyers hieraan vir maande of jare sonder dat hulle toestand gediagnoseer word—dikwels in 'n sanatorium vir tuberkulose. Die gewone tekens wat aan die dag gelê word sluit in huid- en longletsels, limfadenopatie, veral by die longopenings, reënboogvliesontsteking, wat deel mag vorm van 'n ‚uveo-parotitis', hepatomegalie en splenomegalie. Diagnostiese maatreëls sluit biopsie van vel, limfkliere en lewer in. Verhoogde serum-globulien is algemeen. X-strale van die hande toon af en toe bewyse van sarkoïëd-infiltrasie. Die welbekende en meer algemene voorkoms is dié van veelvuldige ‚uitgedrukte' areas in die klein beentjies, soms lyk sommige van die vingerbeentjies verdik, grof gemerk deur trabekels en gedemineraliseerd. Alhoewel daar dikwels daarna gesoek word, is benige letsels beslis ongewoon.

'n Diagnostiese metode waarvan die waarde nie genoegsaam waardeer word nie, is die eenvoudige bepaling van die kalsium in die serum of urien. Abnormaal hoë kalsiumwaardes word baie meer algemeen aangetref as die skeletletsels hierbo genoem. Die minste wat gedoen behoort te word, is die eenvoudige Sulkowitch-toets op 'n vroeë-môre urienmonster. Hierdie toets is in 'n redaksionele artikel in hierdie *Tydskrif*[1] bespreek en word gedoen deur gelyke hoeveelhede urien en Salkowitch-reagens in 'n toetsbuisie te meng, wat dan vir 3 minute gelaat word. As daar aan die einde van die tydperk 'n digte newel (van kalsium-oksalaat) in die vloeistof voorkom, dan is daar waarskynlik 'n oormatige urinêre kalsiumuitskeiding. Die abnormaliteit van kalsiummetabolisme kan dan vasgestel word deur die berekening van die serumvlak en van die kalsiuminhoud van 'n 24-uur-versameling van urien.

As gevolg van langdurige hiperkalsemie en hiperkalsiurie mag kalsium in die nierparenchiem gedeponeer word (nefrokalsinose) en nierstene mag gevorm word. Ernstige nierbeskadiging word veroorsaak en dan dreig die dood weens uremie. Die ander ernstige bedreiging staan in verband met die oë. Kalkagtige neerslae mag in die horingvlies (‚bandkeratopatie'), bindvlies en sklera voorkom, met gevolglike gesigsverlies.

Davidson *et al.*[2] het verdere gevolge weens die hiperkalsemiese toestand aangemeld, naamlik peri-artikulêre sagte-weefsel verkalkings by die skouer en heupgewrigte en voortydige slagaarverkalking by jongmense. Scholz en Keating,[3] van die Mayo-Kliniek, het onlangs die nierverstoring by sarkoïdose bespreek. Beide groepe skrywers het daarop gewys dat, alhoewel granuloomagtige sarkoïedneerslae in werklikheid in die niere mag plaasvind, is die neerslag weens kalsium die hoofbron van beskadiging. Aan

the kidneys, the major source of damage is from the deposition of calcium. On the other hand, Berger and Relman[4] have reported upon a patient with renal impairment in whom sarcoid lesions were found in the kidney by renal biopsy and in whom there was neither hypercalcaemia nor nephrocalcinosis. The calcium output in the urine has been less frequently determined than the serum-calcium level, but in one of Scholz and Keating's cases it is noteworthy that the 24-hour urine figure was around 400 mg. with a serum level of only 10·8 mg. per 100 ml.

In patients who present with the complication of disordered calcium metabolism the most important differential diagnosis is from hyperparathyroidism. The latter condition, which most commonly presents with renal calculi or renal impairment without bone disease, may closely mimic sarcoidosis clinically. Usually one associates hyperparathyroidism with a lowered level of serum inorganic phosphorus, but if renal failure has supervened this may be normal or raised, as in sarcoid. In such a case, therefore, every effort mut be made to exclude sarcoidosis by the use of the diagnostic measures mentioned above.

The actual causes of the disordered calcium metabolism in sarcoidosis are somewhat difficult to determine. Although the obvious source is the skeleton, yet apart from minor localized infiltrations, generalized osseous involvement or rarefaction is not found. Hyperparathyroidism has to be considered as a possible cause. There is, however, no evidence of this condition in sarcoidosis; the serum phosphorus is never lowered, the parathyroid glands are not pathologically abnormal, and osteitis fibrosa does not occur. The hypercalcaemia cannot be accounted for by a raised serum protein. For one thing the protein (globulin) increment is not sufficient, and secondly the important rise in serum calcium takes place in the filterable fraction. It has recently been shown independently by Albright's and Dent's groups,[5, 6] that the disorder of calcium metabolism includes a remarkable over-absorption of calcium from the bowel, with abnormally low faecal calcium and inversion of the usual ratio of faecal to urinary calcium. Both workers remark on the similarity of this to the effect of overdosage with vitamin D. Albright's group then suggests that some abnormal calciferol-like substance may be formed in sarcoid, while Dent postulates an excessive sensitivity to vitamin D. The over-absorption of ingested calcium, however, cannot be the whole story, because balance studies have indicated that the urinary output of calcium may actually be greater than the total intake by the mouth. Furthermore, it cannot satisfactorily explain those cases of hypercalciuria without hypercalcaemia. There is still much about this strange facet of an odd disorder that we do not understand.

1. Editorial (1955): S. Afr. Med. J., **29,** 1247.
2. Davidson, C. N., Dennis, J. M., McNinch, E. R., Willson, J. K. V. and Brown, W. H. (1954): Radiology, **62,** 203.
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4. Berger, K. W. and Relman, A. S. (1955): New Engl. J. Med., **252,** 44.
5. Henneman, P. H., Dempsey, E. F., Carroll, E. L. and Albright, F. (1956): J. Clin. Invest., **35,** 1229.
6. Anderson, J., Harper, C., Dent, C. F. and Philpot, G. R. (1954): Lancet, **2,** 720.

die ander kant het Berger en Relman[4] verslag gelewer oor 'n pasiënt met nierbelemmering by wie sarkoïedletsels in die niere deur middel van nierbiopsie gevind is en by wie daar nòg hiperkalsemie, nòg nefrokalsinose was. Die kalsiumproduksie in die urien is minder dikwels bepaal as die serumkalsiumvlak, maar by een van Scholz en Keating se gevalle is dit opmerkingswaardig dat die 24-uur-uriensyfer om en by 400 mg. was, met 'n serumvlak van slegs 10·8 ml. per 100 ml.

By pasiënte wat die komplikasie van versteurde kalsiummetabolisme toon, is oormatige byskildklierwerking die belangrikste toestand waarvan daar by onderskeidende diagnose onderskei moet word. Laasgenoemde toestand, wat meestal nierstene of nierbelemmering sonder siekte van die beenstelsel toon, mag klinies amper presies soos sarkoïdose lyk. Gewoonlik assosieer 'n mens oormatige byskildklierwerking met 'n verlaagde vlak van anorganiese serum-fosfor, maar as nierondoeltreffendheid tussenby getree het, mag dit, soos by sarkoïed, normaal of verhoog wees. By sò 'n geval moet daar dus alle pogings aangewend word om sarkoïdose uit te sluit deur gebruik te maak van die diagnostiese maatreëls hierbo genoem.

Die werklike oorsake van die versteurde kalsiummetabolisme by sarkoïdose is ietwat moeilik om vas te stel. Alhoewel die skelet die klaarblyklike oorsprong is, word daar tog, behalwe kleinere plaaslike infiltrasies, nie verspreide benige aantasting of verdunning aangetref nie. Oormatige byskildklierwerking moet as 'n moontlike oorsaak oorweeg word. Daar is egter geen bewys van hierdie toestand by sarkoïdose nie: die serum-fosfor word nooit laer nie, die byskildkliere is patologies nie abnormaal nie en osteitis fibrosa kom nie voor nie. Die hiperkalsemie kan nie deur 'n verhoogde serum-proteïen verklaar word nie. Eerstens is die proteïen- (globulien) vermeerdering nie voldoende nie en tweedens vind die belangrike verhoging van serumkalsium in die filtreerbare deel plaas. Onafhanklik van mekaar, het Albright en Dent se groepe,[5, 6] onlangs getoon dat die verstoring van kalsiummetabolisme 'n merkwaardige oor-absorbering van kalsium vanuit die ingewande insluit, met abnormaal lae ontlastings- en urinêre kalsium. Beide navorsers maak aanmerking van die enersheid hiervan en die uitwerking van té groot dosisse van vitamien-D. Albright se groep stel toe voor dat een of ander abnormale kalsiferolagtige bestanddeel by sarkoïdose gevorm mag word, terwyl Dent op 'n oormatige sensitiwiteit tot vitamien-D aanspraak maak. Die oor-absorbering van ingenome kalsium kan egter nie die hele saak verklaar nie, aangesien balansstudies aangedui het dat die urinêre produksie van kalsium in werklikheid groter mag wees as die algehele inname per mond. Vervolgens, dit kan nie die gevalle van hiperkalsiurie sonder hiperkalsemie verklaar nie. Daar is nog heelwat aangaande hierdie eienaardige faset van 'n sonderlinge siekte wat ons nie verstaan nie.

1. Van die Redaksie (1955): S. Afr. T. Geneesk., **29,** 1247.
2. Davidson, C. N., Dennis, J. M., McNinch, E. R., Willson, J. K. V. en Brown, W. H. (1954): Radiology, **62,** 203.
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RESULTS OF OUT-PATIENT TREATMENT OF INFANTILE GASTRO-ENTERITIS

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Gastro-enteritis was certified as the cause of 721 deaths in Cape Town† in the year ended 30 June 1955.[1] Thus, with 'arteriosclerotic and degenerative heart disease'—which caused 724 deaths—it headed the list of causes of death in the city. The brunt of the mortality from gastro-enteritis falls on non-European infants. In 1954-55 the total deaths in non-Europeans from birth to 1 year from all causes (including birth trauma, prematurity, etc.) was 1,069. Of these, 481 (45%) were due to gastro-enteritis. In the same year the total deaths in European infants from birth to 1 year were 72, and only 6 of these were due to gastro-enteritis.

No figures are available for the incidence of gastro-enteritis in Cape Town but it is probably fair to take it as at least 10 times the number of deaths, i.e. 7,000 cases per year. Clearly it is not possible to admit such numbers to hospital and the majority must be treated at home or as hospital out-patients. Although no count is kept of the thousands of cases of gastro-enteritis treated at Groote Schuur Hospital each year, an idea of the number of dehydrated cases may be obtained from the figures for subcutaneous infusions given to out-patients. These were 1,027 in 1954, 1,863 in 1955, and 1,013 from January to June 1956.

It has been found best to lay down a standard regime of treatment for out-patients. The principles of the treatment are described elsewhere (p. 452) in this issue.[2] The results have been uncertain because about half the cases are only seen once or twice while sick, and then never return to hospital. The work reported here was therefore undertaken to test the efficacy of out-patient treatment by thorough follow-up in an unselected sample. This consisted of 100 consecutive infants presenting at the casualty department of the hospital with gastro-enteritis between May and July 1955.

A broad definition of gastro-enteritis was used. All cases presented with significant diarrhoea as a main symptom and in 75 it was accompanied by vomiting. In many of them other pathological conditions were present initially or became manifest later. These were included for the following reasons:

1. Even if there is parenteral disease, diarrhoea may still be due to pathogenic organisms in the bowel.

2. Prolonged diarrhoea may itself cause malnutrition or susceptibility to parenteral infection.

3. The underlying cause of diarrhoea (e.g. measles or kwashiorkor) may not be recognizable at the first visit, which is in some cases the only visit.

4. Treatment of diarrhoea follows the same principles whether there is associated disease or not.

All the patients were non-European and the only selection was to limit the series to those not more than 2 years old. The criterion for out-patient treatment was simply the availability of cots, and only 6 could be admitted. All out-patients were examined and treated by the writer.

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† These figures are for the Cape Town Municipal area, which does not include most of the northern suburbs, where there is also a considerable mortality from gastro-enteritis.

CLINICAL OBSERVATIONS

The age distribution of the children is shown in the graph in Fig. 1. The maximum incidence was at 3 months; 77% were under 12 months old. There were 45 males and 55 females; 58 were Coloured, 32 African and 10 Malay.

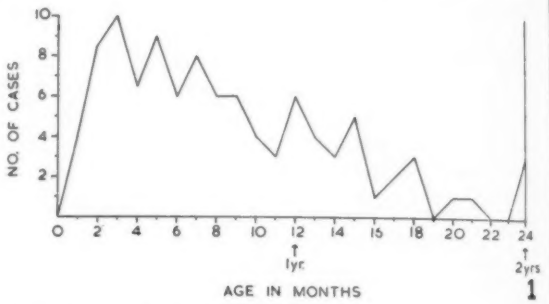

Fig. 1. Age distribution of cases in this series.

The feeding regime before illness is recorded in Table I.

TABLE I

Feeding History	*Cases*
Breast only	2
Breast and Bottle	8
Breast and Food	6
Bottle only	39
Food only	29
Unrecorded	15
	100

The clinical findings are indicated briefly in Tables II, III and IV, in which a differentiation is made between those

TABLE II

Ultimate Clinical Diagnosis	*Mild Cases*	*Severe Cases*
Pure gastro-enteritis	13	40
Gastro-enteritis and severe conjunctivitis (1) or salivary-gland infection (1)	—	2
Gastro-enteritis and upper-respiratory-tract infection	10	12
Predominant upper respiratory infection and mild gastro-enteritis	7	—
Gastro-enteritis and otitis media	—	2
Kwashiorkor and upper respiratory infection	—	2
Kwashiorkor	2	4
Severe parenteral infection—tuberculosis of lung (1), bronchopneumonia (3), measles (1)	—	5
Numerous roundworms	—	1
	32	68

infants who were considered to be suffering from a relatively mild degree of illness and those who were seriously ill. The main criterion for this division was the clinical assessment of the degree of dehydration. This was based primarily on examination. Dryness and loss of resilience of the skin; sunken, dry eyes with reduction of eyeball tension, sunken fontanelle, and dry mouth, are the most specific signs but each must be interpreted with caution. The skin is sometimes lax from undernutrition alone, when there is great reduction of subcutaneous fat. On the other hand loss of resilience is often difficult to detect in a plump baby with abundant subcutaneous fat but recent acute loss of fluid. Here the characteristic 'doughy' feel of the subcutaneous tissue can only be learnt by experience. The eyes may also be sunken in gross undernutrition, sticky eyes may be due to infection, and eyeball tension is difficult to assess. In older babies the fontanelle is either completely closed or so small that a finger cannot be got right into it, and if the baby is crying or has meningitis a patent fontanelle may not be sunken even in a dehydrated infant. The mouth may be dry from hyperpnoea.

TABLE III

*Nutrition**	*Mild Cases*	*Severe Cases*
Good nutrition	11	4
Adequate nutrition	19	35
Moderate undernutrition	1	20
Gross undernutrition †	1	9
	32	68

* Weight for age.
† The weights are shown in Table IV.

TABLE IV. WEIGHTS OF THE GROSSLY UNDERNOURISHED INFANTS

Case No.	*Weight (lb.)*	*Age*	*Before or after Treatment*
14	3½	6 weeks	before
7	4½	2½ months	before
65	6½	3 months	before
1	7½	4 months	before
86	7½	5 months	after
71	6¾	6 months	before
61	9½	8 months	after
29	10½	1 year	after
28	10½	13 months	after
11	10	18 months	before

With *severe* dehydration, in addition to these signs the infant looked ill, was apathetic and weak, and often showed signs of circulatory collapse or acidotic breathing.

In 26 instances it was later possible to check the original clinical assessment by calculating the percentage dehydration from the gain in weight during the first week of treatment in cases that recovered and never showed oedema. The figures for these are shown in Table V. No allowance was made for increase of weight from tissue growth in this first week, because diarrhoea usually continued for several days and none but the mildest cases were back on full-strength milk before the 4th day.

All but one of the 22 clinically dehydrated cases in Table V were, by calculation, at least 5% dehydrated. The calculated degrees of dehydration ranged up to 17%. Only in 4 cases were they significantly less than the clinical estimate, and in these the diarrhoea had not completely stopped at the second weighing. Thus the table serves as a rough sample of the reliability of the clinical assessment of dehydration. On the other hand, the 4 cases from the group not clinically dehydrated are too few to be representative. The rest could not be used because they did not return in a week or were not weighed twice. The 4 examples that do appear in the table, however, suggest that some of the mild cases were more dehydrated than was suspected clinically.

TABLE V. CLINICAL ASSESSMENT COMPARED WITH DEGREE OF HYDRATION CALCULATED FROM WEIGHT GAIN ON RECOVERY

Case No.	*(1) Weight before Treatment (lb.)*	*(2) Weight after Treatment (lb.)*	*Days between (1) and (2)*	*Retrospective Calculated % Dehydration (2)—(1)/(2)*	*Clinical Assessment of Dehydration at first visit*	*Fluid given in 1st Subcut % body weight*
Clinically Dehydrated						
18	12½	14½	4	14%	mild	2½%
23	9½	11½	5	14%	moderate	5%
26	8½	9½†	2	7%	severe	10%
38	9½	10½	7	10%	mild	2½%
40	12½	14½	6	13%	moderate	5%
41	6	6½	5	8%	mild	2½%
43	18½	19½*	4	2·5%	moderate	5%
44	12½	15	5	17%	severe	10%
45	6	7	3	14%	severe	10%
51	18	21½	7	16%	considerable	7½%
68	14½	16½	7	9%	moderate	5%
75	11½	12⅝†	2	8%	severe	10%
79	15	16½	6	9%	considerable	7½%
82	13½	15 †	3	8%	considerable	7½%
84	7½	7½	1	6·5%	moderate	5%
87	6½	7	7	12·5%	moderate	5%
88	10½	12½	5	12%	moderate	5%
89	10	11½	1	15%	considerable	7½%
90	5½	6½	6	10%	severe	10%
91	15½	16¾	2	7%	mild	2½%
96	14	15½	4	10%	moderate	5%
98	13½	14½†	2	5%	considerable	7½%
				Average 10·2%		Average 6·4%
Not Clinically Dehydrated						
58	13½	14½	5	6%	not	
60	17½	19 †	6	8%	not	
77	13	13½	4	2%	not	
94	16½	17½	5	4%	not	

* = diarrhoea better but not completely cleared at 2nd weight.
† = very mild case, on full strength milk from 2nd day. Part of weight gain probably due to tissue growth.

Rectal swabs were only taken where there was a dysenteric stool, lack of response to treatment, or a history of previous gastro-enteritis with specific pathogens in the stool. They were only cultured for Salmonella and Shigella. Of 12 such cultures, 6 showed *Shigella flexneri* and 2 showed *Salmonella* spp.; in one instance both organisms were found. There was a history of Ascaris infestation in 4 children but in only 1 did this seem to be related to the diarrhoea.

TREATMENT

Three cases were admitted at their first visit. The rest were treated as out-patients but 3 of them were subsequently admitted because they were not responding. This left 94 treated entirely as out-patients.

1. In the out-patient department a subcutaneous infusion of electrolyte solution was given to all cases that were clinically assessed as dehydrated. Both dehydrated and mild cases were then sent home, usually on the following regime:

2. Feeds: 1st day, oral Darrow's solution only; 2nd day half-strength milk only; 3rd day, 3/4th-strength milk only; 4th day, full-strength milk only; 5th day onwards, gradually back to solid food (if the baby was weaned).

3. Oral sulphadiazine, given for 5 days at 1½ gr. per lb. per day (either a chocolate-flavoured mixture containing 4 gr. per teaspoonful or the standard 0·5 g. tablets were used, depending on the infant's age). Treatment was started with chloramphenicol or a tetracycline if sulphonamide had been adequately tried outside hospital without success. Five mild cases were treated without antibiotics.

The mothers were given a printed sheet of instructions, told about the necessity for cleanliness in the preparation of feeds, and asked to return in 1-3 days, depending on the severity of the case. They were warned that the child might

giving an adequate amount of subcutaneous fluid need not exceed half an hour.

Normal saline and Darrow's solution were the only fluids used subcutaneously in this series. Generally, about half the fluid was given as normal saline and half as Darrow's solution. The saline was usually given first. In acutely ill or shocked patients more saline (or even all saline) was given, while for chronic, weak patients more Darrow's (or even all Darrow's) was given. (Note: It is now recommended that some of the fluid given subcutaneously should be N/2 saline or half-strength Darrow's solution with 2½% dextrose.[2]

TABLE VI

Visit No.	Total No. of patients seen	Died while in O.P. dept. before or after subcut	Admitted at this visit	Treated as out-patient	Given a subcut at this visit	Feed ordered for 1st 24 hours after present visit		Cumulative total nos. of O.Ps. in the 3 different antibiotic groups at each stage		
						Darrow's solution	Milk	Sulphadiazine only	Tetracycline group	Chloramphenicol
1	100	1	3	96	64	81	15	87	2	2
2	76	1	1	74	16	3	71	81	5	4
3	44	1	—	43	4	1	42	78	6	6
4	21	—	2	19	1	—	19	—	—	—
5	8	—	—	8	—	—	—	77	7	6
6	6	—	—	6	—	—	—	—	—	—
7	3	—	—	3	—	—	—	—	—	—
8	2	—	—	2	—	—	—	—	—	—
9	1	—	—	1	—	—	—	—	—	—
10	1	—	—	1	—	—	—	—	—	—

deteriorate at any time and if so must be brought back immediately. The standard regime was varied to suit the individual case. Associated disease was given appropriate treatment. At return visits, further 'subcuts' were given if dehydration was still present, the feeds were adjusted if necessary, and chloramphenicol or a tetracycline was substituted for the sulphadiazine if there was no improvement in a severe case after 48 hours. The treatment actually given is shown in Table VI.

Subcutaneous Infusions were given on the indications and by the technique described elsewhere (p. 453) in this issue.[2] As the patients were sent home after being seen and a 'subcut' is not a dangerous procedure, this rule was used: When in doubt whether a baby might be dehydrated or develop dehydration soon, give a 'subcut'.

Dehydration was assessed as mild, moderate, considerable or severe. For mild dehydration, less than 5% of the baby's body-weight was given in fluid, for moderate dehydration 5%, for considerable dehydration 7½% and for severe dehydration 10%. For example, a 10 lb. (5 kg.) baby severely dehydrated was given 10% of 5 kg. of fluid=500 ml. The calculation was based on weights before treatment and, for simplicity, the approximation 1 lb.=½ kg. was used.

The total amount of fluid given by 'subcut' ranged in this series from 150 to 600 ml., with an average between 350 and 400 ml. It was found difficult to get much more than 300 ml. into one site (i.e. 600-700 ml. into paired sites). This means that should an infant weighing over 12 lb. be severely dehydrated it would not be possible to give 10% of its body weight in fluid unless 3 sites were used. However, this difficulty was not experienced because only 2 of the 46 children weighing over 12 lb. showed severe dehydration. The two each weighed only a little over 12 lb. and both did well on 600 ml. of subcutaneous fluid. The time involved in

Follow Up

The mothers were encouraged to bring their babies back until they had completely recovered. Of the 94 out-patients,

TABLE VII. FOLLOW UP

Result observed	49
Responded to 1st or 2nd letter	17
Successful home visit	9
Through referring GP, local clinic, or parent's place of work	5
Search of Cape Town and Bellville death registers for 1955	2
Returned to hospital for another illness in next 1½ years	6
Untraced	6
	94

49 made sufficient return visits for the results of treatment to be clearly seen (Fig. VII). Further follow-up was difficult, as can be appreciated from the fact that letters to 18 were twice returned undelivered by the Post Office, the addresses being of very poor homes which had no street numbers. The 6 which were never traced all fell into this problem group.

Results of Treatment

The 6 children who were admitted are not considered further. Of the 88 out-patients who were traced, 76 recovered and 12 died (Table VIII). Early in 1957, the indices to the death registers in the offices of the District Registrar at Cape Town and Bellville, and the Native Commissioner for Cape Town, were searched for the 6 untraced cases (see Table VIII) and also the 12 known out-patient deaths. The records of all the 12 known deaths were easily found but there was no entry of any of the untraced infants for the year 1955 (i.e. for 5-6 months after the completion of treat-

TABLE VIII

	Out-patients throughout	*Admitted at 1st visit*	*Abmitted later*	*Total*
Not traced	6	—	—	6
Result known ..	88	3	3	81
Recovered	76	3	3	81
Died*: Early ..	8	—	1	9
Late ..	4	—	—	4
Total Cases ..	94	3	3	100

* Early=within 5 days of last visit.
Late=2-6 weeks after last visit.

ment). This is very strong, though indirect, evidence that all of these patients survived the gastro-enteritis for which they were treated. Taking these as survivals, the mortality rate in the children treated solely as out-patients was 12 out of 94, or 13%. All 12 deaths were among the 62 cases classified as severe (mortality 19%). There was no death among the 32 mild cases. Three of the untraced cases were in the severe group and 3 in the mild group.

Adverse Effects of Treatment: Only one child developed a rash attributable to sulphadiazine and no haematuria or anuria was observed in this series. Although a total of 85 'subcuts' (involving 190 puncture sites) was given to 64 patients, local infection was not seen. This does sometimes occur; the author has seen bilateral cellulitis of the chest wall follow a 'subcut'. It is clearly necessary to use a careful aseptic technique, for these infants are debilitated and specially vulnerable. Local dissemination of infection at the injection sites is likely to be promoted by the use of hyaluronidase, which is part of the technique. One baby died ½ hour after a 'subcut'. She had very severe gastro-enteritis and should have been admitted. Death was probably inevitable as the result of irreversible shock which could not be corrected by subcutaneous infusion. However, the slight impairment of ventilation from the weight and rigidity of 100-300 ml. of fluid on each side of a small infant's chest might be critical in the presence of pneumonia or acidotic breathing. It may be safer to use the anterior thighs for 'subcuts' in such patients.

Darrow's solution was given first or alone in 21 'subcuts'; 3 of the early deaths occurred after these (14%). Saline was given first or alone in 61 'subcuts'; 5 of the early deaths followed these (8%). The difference between the incidence of early deaths after the 2 types of infusions is not statistically significant ($\chi^2=0{\cdot}648$, n=1, P=0·3) and Darrow's solution has the theoretical advantage that it replaces some of the potassium lost in the stools and vomitus.

NOTE ON THE RELATION OF KWASHIORKOR TO GASTRO-ENTERITIS

As indicated in Table II, there were 8 definite cases of kwashiorkor in this series. Several other cases showed signs of protein deficiency but not the complete syndrome (e.g. protein-deficient diet, failure to gain weight, lack of energy, and the typical skin or hair changes, but no oedema). In some of these the diagnosis of 'pre-kwashiorkor' was confirmed by subsequent development of oedema while the child was under observation. Most of the infants with kwashiorkor were over 1 year old; in fact 10 of the 22 cases in children over 1 year of age gave evidence of kwashiorkor (6) or pre-kwashiorkor.

In the 8 definite cases of kwashiorkor, 3 already showed oedema when they presented with diarrhoea, but it had appeared from 3 days to 3 weeks after the start of the diarrhoea. In 4 others there was no oedema when the infant presented with gastro-enteritis and dehydration but, on rehydration and recovery, oedema appeared. Other features of kwashiorkor were present and the urine was normal. The 8th case had been in hospital 3 months previously with nutritional oedema. After discharge she had received a moderate amount of milk and remained well until 4 days before her return, when oedema reappeared at the same time as the diarrhoea started. Serum albumin was 3·2 and globulin 2·0 g. per 100 ml. and when the diarrhoea subsided, on treatment with sulphadiazine and Eledon, the oedema disappeared.

In all these children diarrhoea appeared before the oedema or at the same time. It was interesting that 4 who seemed at first to have gastro-enteritis, with dehydration and some stigmata of protein deficiency, really had full-blown kwashiorkor and their oedema became unmasked (or even precipitated) by parenteral rehydration. Two further cases which never showed skin or hair changes developed transient mild oedema the day after subcutaneous infusions. Their diets were deficient in protein and it was thought that protein loss from acute gastro-enteritis may have determined their tendency to oedema.

DISCUSSION

Aetiology

The bacteriology of the stools was studied in only 12 cases. *Shigella flexneri* was cultured from 6 and would presumably have been found in more had all the stools been examined. Kahn[3] at Baragwanath found the incidence of Shigella infection in out-patients to be 28% in summer and 12% in winter. He also found Salmonella in 19% in summer and 4% in winter.

In a series similar to the present one, of out-patients with 'gastro-enteritis' at Groote Schuur Hospital, Woolf and Kipps,[4] later in 1955, studied rectal swabs for *E.coli*. They found 0111, 055, 0119 or 0128 in only 6 of 96 cases. On the other hand, Coetzee and Pretorius[5] found these four *E.coli* strains in 8 out of 69 control out-patients without gastro-enteritis in Pretoria, so that they do not appear to play much part in the type of gastro-enteritis under consideration in this paper.

The incidence of upper respiratory infection in the present series was 31%; it was higher in the mild cases (53%) than the severe (21%.) Kahn[3] found that upper respiratory infection was associated with 32% of cases of gastro-enteritis in the summer but 82% in the winter.

Kwashiorkor was seen in 8 cases in the present series and evidence suggesting protein deficiency in several more. Diarrhoea preceded oedema in most of them and it is clear that once a protein-deficient weanling develops diarrhoea, whatever its pathogenesis, a vicious cycle can be established. In addition, many infants with subacute gastro-enteritis who come to Groote Schuur Hospital have been given only glucose water or barley water for a week or more and must have become very depleted in protein. To avoid this, the treatment regime used introduced half-strength milk on the 2nd day and aims at reaching full-strength milk by the 4th day.

Only 2 infants in the series were entirely breast-fed and the low incidence of gastro-enteritis under 3 months of age may be related to more extensive breast-feeding of non-European babies in the first two or three months of life. The home visits made by the author left very little doubt that many of the children lived in poor, overcrowded homes where adequate sterilization of artificial feeds must be very difficult to achieve. Observations in this series fully supported the general view that the main underlying cause of gastro-enteritis is poor feeding hygiene.[6]

Recurrences. The incidence of recurrences was not deliberately studied. However, in taking the histories it was noted that 22 children had had one or more previous attacks of gastro-enteritis and 6 others had gastro-enteritis at least a month before coming to hospital. The current attack may have represented reinfections. Furthermore, during the follow-up work (which covered less than half the cases) 10 infants were found to have had one or more recurrence, 1-12 months after the treatment described in this article. The frequency of recurrences points to the persistence of the poor feeding-hygiene in many cases.

Recurrences also appeared to be important as a causal factor in undernutrition. Twelve (71%) of the 17 definitely undernourished children over 5 months of age had had previous attacks of gastro-enteritis, compared with only 9 (19%) of the 47 adequately nourished of the same age-group. Another explanation of these figures may be that the babies who were given insufficient food were also given it less hygienically. The incidence of undernutrition in the series was 31 (i.e. 31%) and this does not include 4 of the kwashiorkor cases. Assessment of undernutrition was necessarily arbitrary because there are no figures available for 'normal' weights of non-European infants at Cape Town. A significant difference was found between the number undernourished in the severe cases (41%) and in the mild group (6%).

Treatment

The most important part of the treatment given was subcutaneous infusion of fluid to all dehydrated cases. The volume of fluid infused was intended to replace fluid loss, and thereafter the daily maintenace of fluid was provided by frequent feeds, first of Darrow's solution and then of milk. The amount given to a severely dehydrated baby in the 'subcuts' was 10% of the body-weight and, for lesser grades of dehydration, proportionately smaller amounts. The average dehydration calculated in retrospect for the 22 clinically dehydrated cases shown in Table V was 10·2% of the body-weight as it was on recovery. The average amount of fluid prescribed for them was 6·4% of the body-weight *before treatment*. But, because 1 kg. was taken as being equal to 2 lb. there is a 10% error and the average should be slightly higher than this figure. Thus in this sample only 63% of the estimated water loss was restored by the initial 'subcut'. All the cases in Table IV must have made up the balance of their fluid loss by oral fluid (or by a subsequent 'subcut' in 5 of the 22). It is, in fact, safer not to replace *all* the fluid loss by a rapidly administered 'subcut' because of the impossibility of determining the exact amount of fluid depletion clinically, and the risk of overloading the circulation. Had the cases in Table V all been given 50% more fluid than they received (making the average amount given about 10% of the hydrated-weight) they might have been overloaded. The amount of fluid actually given appears to have been safe from this point of view.

In treating dehydrated gastro-enteritis, correction of dehydration is more important than chemotherapy. However, in treatment trials[7,8] there have always been better results in the groups on chemotherapy than in controls without it, and no recent workers have felt justified in withholding chemotherapy in the control cases that deteriorated. In the present series sulphadiazine was used as the standard anti-bacterial agent, in a dosage of 1½ gr. (0·1 g.) per lb. per day. In the British M.R.C. trials[7] results were rather better with sulphadiazine (2 gr. per lb. per day) than with aureomycin or chloramphenicol. Theoretically to combat any parenteral infection present, a soluble sulphonamide should be better than one which is poorly absorbed. MacDougall[9] found that sulphadimidine—another soluble sulphonamide—was as good as phthalylsulphathiazole or sulphaguanidine for pure infantile gastro-enteritis and better where there was parenteral infection. Salmonella and Shigella infections were excluded from the M.R.C. trials, but Stein[8] found little difference between the results with sulphonamide, terramycin or chloramphenicol in Shigella and Salmonella gastro-enteritis.

Results

The 13% mortality found in this series is lower than had been expected and is only a third of the in-patient mortality (40-50%) in Western countries a quarter of a century ago.[10] The results are worse than those at present obtainable in Western countries overseas and in European patients in South Africa. For example, the mortality was 3% in the British M.R.C. trials in 1953[7] and 2% among European in-patients at Groote Schuur Hospital in 1954.[11] In these groups all dehydrated cases could be admitted for intravenous therapy and the proportion of severe cases was lower; it was 35% in the M.R.C. series. On the other hand the results in the present series are better than those for non-European in-patients in this country, in whom mortality figures like 25%[8] and 44%[11] are being obtained. However, non-European cases that are admitted are a more severe group and many are moribund.

No one factor can be found common to all the 12 out-patient deaths in this series. In the 4 cases that were not brought back and died at home (the *late deaths*) maternal mismanagement seems to have been the main reason for death. All 4 showed some manifestation of malnutrition yet none was very severe (as indicated by the length of their survival without treatment). The mother of one could not speak English or Afrikaans and could not count. The mother of another refused admission of her baby for kwashiorkor.

In the 8 *early deaths* 5 were malnourished (4 were undernourished and 1 had kwashiorkor) and had, with one exception, had diarrhoea for at least a week. The other 3, with adequate nutrition, presented with an acute illness of 1-3 days duration. Associated disease was important in only 1 case, with pulmonary tuberculosis. Four cases showed severe, 1 considerable, 2 moderate and 1 slight dehydration. There was some preponderance of the younger cases in the group that died; 5 of the 8 were 5 months old or less and only 1 was over a year old. All were treated with sulphadiazine and all but one received one or more 'subcut'.

Clinical assessment of severity was found to be reliable in the mild cases, all of which remained mild. The severe ones

were assessed as very severe or moderately severe, and admission was sought for all the former. Fifteen cases were considered very severe; 5 of them were admitted sooner or later, of whom 1 died, and 6 died as out-patients. On the other hand there were 6 deaths among the 52 out-patients assessed as moderately severe. Two of these were late deaths and can be attributed to failure of the mother to complete the treatment. Of the 4 early deaths in these cases where admission was not considered essential, 3 had acute pure gastro-enteritis and 1 was a case of kwashiorkor. It is the experience of the Kwashiorkor Research Unit, Cape Town, that kwashiorkor is a treacherous disease and that sometimes children die suddenly when they appear to be progressing favourably.[12] It appeared from the present series that acute gastro-enteritis with dehydration is similarly treacherous.

Although the mortality in the cases assessed as moderately severe was only 6 out of 52 (11%), no means was found of telling which cases of acute gastro-enteritis or kwashiorkor would suddenly deteriorate. Out-patient treatment gave satisfactory results in mild gastro-enteritis and moderately severe chronic gastro-enteritis where the mother could cooperate. Nevertheless, there were deaths in cases with dehydrated acute gastro-enteritis, very severe chronic gastro-enteritis, kwashiorkor and those where the mother was unable to look after her child properly. Ideally all these should have been admitted to hospital and the treatment used in this series was only justified by the impossibility of admitting the cases.

Treatment of this series was uncontrolled. However, the sample appears to have been fairly representative of the disease pictures seen throughout the year, though possibly more virulent gastro-enteritis may be seen in the summer. It is hoped that the present results may be of use in evaluating future modification and improvements in treatment.

Comment

Many of the mothers of babies seen in this series had stopped breast-feeding prematurely. This may be unavoidable if the mother has to work away from her child, but artificial milk mixtures were clearly being given unhygienically and often in inadequate amounts. Babies were often given excessively dilute milk feeds and weaned babies no milk at all. It is felt that not all these faults were due to poverty and that, with universal education of mothers in the principles of infant feeding, gastro-enteritis as well as marasmus and kwashiorkor would become as infrequent as they have in Western Europe and North America. Instruction on infant feeding is one of the main functions of the infant welfare clinics and one of the big difficulties is the considerable number of poor mothers who either do not know of these clinics or do not choose to attend them. Perhaps the clinic workers should visit the homes of mothers who do not attend, and infant feeding might be taught in all girls' schools.

Many of the mothers seen in this study had to travel long distances with frail sick babies, sometimes in the rain, to reach hospital. Their fares were considerable and often several visits were necessary. At the hospital there was often a long wait because of the enormous case-load. These are the reasons why mothers fail to return for continuation of treatment, and there is another facet to the problem—that the mother's other children have to be neglected or some arrangement made for their care in her absence. The care of the patients described in this paper, with the exception of a few who needed X-rays, night emergencies, etc., could have been carried out in peripheral clinics nearer the baby's home. It would also seem desirable for treatment of sick babies to be conducted by those who instruct on and supervise their care in health. These considerations point to the need for closer integration between the hospitals and the infant welfare clinics and extension of the facilities at these clinics to include treatment.

SUMMARY

Gastro-enteritis accounts for nearly half the total deaths of non-European infants under 1 year of age in Cape Town. Because of the high incidence of the disease only a very small proportion of patients can be admitted to hospital for treatment.

Most dehydrated cases are at present treated at hospital out-patient departments. The most important part of this treatment is subcutaneous infusion of electrolyte solutions to replace fluid loss. The result of this out-patient treatment has been uncertain because many of the mothers failed to bring their infants back for further treatment or follow-up.

One hundred consecutive cases presenting with diarrhoea were therefore studied in the out-patient department at Groote Schuur Hospital, Cape Town. The clinical findings and incidence of parenteral infection and malnutrition are described. Of these, 94 were treated throughout as out-patients. Treatment was with a standard regime and uncontrolled; 64 patients were given one or more subcutaneous infusions. Some practical aspects of the treatment given are reviewed.

After a difficult follow-up the mortality for the out-patients was found to be 13%. Deaths were confined to the clinically dehydrated cases and there were no deaths in the cases originally assessed as mild.

The results of this study and possible reasons for the deaths are discussed. It is suggested that extension of treatment facilities to peripheral clinics would be advantageous to the mothers and babies, and reduce the case-load on hospitals.

I wish to thank Prof. F. J. Ford and Dr. J. D. L. Hansen for their stimulus and advice, and Dr. N. H. G. Cloete, Medical Superintendent of Groote Schuur Hospital, for permission to publish these cases.

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THE OUT-PATIENT OR HOME TREATMENT OF SEVERE GASTRO-ENTERITIS WITH DEHYDRATION IN INFANTS

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It is estimated from one centre in South Africa that 10% of African children succumb to diarrhoeal disorders during the first year of life.[1] This appalling mortality rate from gastro-enteritis is not confined to one city[2] and similar rates are probably applicable in many urban and rural areas in this country. Very many of the deaths can be attributed to dehydration and electrolyte disturbances, with or without the aid of other potentially lethal complications such as broncho-pneumonia or septicaemia. Treatment of gastro-enteritis should, therefore, be directed in the *first place* towards the correction and prevention of water and salt losses. Every other measure used in treatment is of secondary importance, because gastro-enteritis is, in general, self-limiting.

The main part of the offensive against dehydration is borne by the most junior section of the medical staff of the hospitals to which, sooner or later, many of these children are sent. The large number of infants affected in the summer months necessitates an out-patient routine which they may safely be left to follow and which, at the same time, will conform to modern requirements. A scheme that has proved *simple* and effective in this unit[2] is described in the hope that it may be useful to others who are still searching for a solution of the many difficulties involved. It is a scheme, moreover, that can be used by general practitioners anywhere, whether in the home, consulting room or dispensary.

ORAL THERAPY

In the majority of cases replacement of fluid and electrolyte loss can be accomplished orally if treatment is started early. It is only where oral therapy fails that parenteral administration becomes necessary.

The first principle to be followed is to stop all milk or other foods for 24 hours. In their place electrolyte solution should be administered in adequate quantities. This should be sufficient (1) to restore fluids and electrolytes already lost and to cover continuing losses, and (2) to provide for maintenance requirements. A method of working out the volume required is as follows:

1. *Replacement* of losses: It is necessary to replace amounts of fluid equivalent to 5 or 10% of the body weight, depending on the degree of dehydration, i.e. 5% in moderate dehydration, 10% in severe dehydration. This is equivalent to approximately 25-50 ml/lb. or 50-100 ml/kg.
2. *Maintenance* requirements: 70 ml (2½oz.)/lb., or 150 ml/kg., per 24 hours.

For example, a 10 lb. baby with severe dehydration will require (using 10% of body weight to calculate replacement):

1. Replacement fluid 10 × 50 ml = 500 ml.
2. Maintenance fluid 10 × 70 ml = 700 ml.

Total 24 hour fluid = 1,200 ml = 2 pints approx. = 4 oz./lb.

For oral therapy the fluids we recommend are:

(1) ¼-strength Darrow's solution with 2½-5% dextrose, or

(2) ¼-½ normal saline with 2 -5% dextrose.

Where saline is used, potassium chloride 1-2 g. per day should be prescribed separately; its value in treatment is now firmly established.[3] The mother can make up an approximation of solution 2 at home quite simply by adding ½ teaspoon of salt and 1½-2 tablespoons sugar or glucose to 1 pint of water. Darrow's solution ought to be available in every dispensary and tablets for making up oral electrolyte solutions containing sodium and potassium salts should soon be available commercially.

We have found it important to tell the mother exactly, in simple terms, how much fluid the baby is to have in 24 hours. The fluid should be administered at least 3-hourly, and preferably more often, both by day and by night.

On this regime the majority of infants stop vomiting and take the fluids very well. If administration by bottle is unsuccessful an attempt should be made to maintain the schedule by spoon-feeding. The necessity of scrupulously clean utensils must be impressed on the mother. If vomiting continues and there is no improvement, recourse must be had either to continuous intragastric drip or parenteral therapy.

It must be stressed that 24 hours on the glucose-electrolyte solution are usually sufficient, and certainly this regime should not be continued for more than 48 hours even if some diarrhoea is still present.

At the end of the 24-hour period milk should replace the electrolyte solution. Full-strength skimmed milk is best but a half-cream milk with or without lactic acid will do. Skimmed milk is chosen because it supplies protein, mineral salts, some carbohydrate, and water in a form recognised by both mother and baby as food. At the same time fat, the part of the milk most difficult to digest in the presence of an acute bowel upset, is eliminated or at least reduced in amount. If neither of these is available, whole cow's milk diluted 1 : 1 with electrolyte solution, or in milder cases with water, is satisfactory. The concentration of milk should be stepped up day by day so that the infant is having whole-milk feeds, without added sugar, within 3-4 days *even if there is still a certain amount of diarrhoea present*. If feeding is withheld until all diarrhoea has ceased malnutrition may supervene. This is of particular importance for infants in the underprivileged section of the community, whose reserves may be insufficient to see them through long periods of dietary restriction. This clinic has seen many cases of kwashiorkor that were apparently precipitated by the inadequate feeding prescribed with the best of intentions for treatment of diarrhoea. It should be pointed out to the mother that loose stools may persist for 1-2 weeks after the acute attack. This need not cause undue concern providing feeds are being taken well and there is no vomiting.[4]

PARENTERAL THERAPY

The decision to use parenteral therapy depends largely on clinical experience. Certain broad indications for its adoption can be stated:

1. Severe clinical dehydration as judged by inelastic skin, especially over the abdomen, sunken eyes and fontanelle, dry mouth and peripheral circulatory collapse.

2. Continued vomiting even after the introduction of oral electrolyte solutions. In this type of case it may be advisable to start parenteral therapy before obvious signs of dehydration appear.

3. A failure to show clinical improvement even in the absence of vomiting after 6-12 hours of oral electrolyte therapy. (Progressive loss of weight is a valuable objective sign.)

Intravenous patenteral therapy is obviously the best if hospital beds and trained personnel are available. It will not be discussed here. In out-patient departments, in the consulting room or in the home, subcutaneous administration of fluids is a very satisfactory resuscitative measure in the treatment of dehydration.

Technique

A needle is inserted subcutaneously into each lower axilla and strapped to the skin (see Fig. 1). The needles are connected to the infusion bottle by means of a Y-tube. Half an ampoule of hyaluronidase is injected through each needle and the infusion allowed to drip in as fast as it will go.

With a suitable arrangement of tables and overhead bars for suspension of bottles, 6 or more infants may be treated simultaneously, which is very convenient in a large casualty department (Fig. 2). Sepsis is negligible if proper aseptic technique, sterile equipment and disinfection of skin with alcohol are used. Fluid can alternatively be injected subcutaneously by means of a large syringe into the back, with 1 c.c. of hyaluronidase as a spreading agent. This is a useful emergency method in the home but is too time-consuming for mass use and, besides, is probably more painful because of the positive pressure likely to be applied.

Fig. 1. A subcutaneous infusion in progress.

Fig. 2. Administration of subcutaneous infusion in a casualty department.

Solutions for Subcutaneous Infusion

I. Isotonic (normal) saline
II. Darrow's solution.
III. $\frac{1}{6}$ molar sodium lactate
} Absorbed rapidly because isotonic.

IV. $\frac{1}{2}$ N. saline with $2\frac{1}{2}\%$ dextrose, i.e. equal parts N. saline and 5% dextrose.

V. $\frac{1}{2}$-strength Darrow's solution with $2\frac{1}{2}\%$ dextrose, i.e. equal parts Darrow's solution and 5% dextrose.

(Electrolyte-free 5% dextrose should never be given subcutaneously, for it is poorly absorbed and causes irritation.)

Dosage. A volume of fluid equivalent to 5% of the body weight (25 c.c. per lb., 50 c.c. per kg.) may safely be given to any child with diarrhoea. In severely dehydrated infants a volume up to 10% of the body weight (50 c.c. per lb., 100 c.c. per kg.) may be given subcutaneously. The first half of the prescribed fluid should be solution I or II. Solution III is used where acidosis, as judged by hyperventilation, is severe. The remainder of the fluids prescribed should be from solutions IV or V. For example, the 10 lb. baby who is severely dehydrated will require 10×50 ml. $= 500$ ml. of fluid. Of this, 250 c.c. will be isotonic saline or Darrow's solution or $\frac{1}{6}$ molar lactate and 250 c.c. $\frac{1}{2}$ normal saline with $2\frac{1}{2}\%$ dextrose or $\frac{1}{2}$-strength Darrow's solution with $2\frac{1}{2}\%$ dextrose. After the 'subcut', which acts as replacement fluid, the babies can be sent home. Oral feeding with electrolyte solution—to supply maintenance requirements—should be commenced after a 3-hour period of 'nil per mouth'. The children should be seen again after 12-24 hours for assessment of their general condition.

The majority are then much improved and taking fluids well by mouth; others need a booster 'subcut'. It is only the very severe case that fails to respond and has to be admitted to hospital for specialized intravenous therapy.

SUBSIDIARY TREATMENT

An antibiotic is recommended for a period of 5 days. The most satisfactory in our experience is (1) sulphadiazine or sulphamezathine (Sulphadimidine), 1½ gr. per lb. per day, or (2) chloromycetin 50 mg. per lb. per day. Sulphonamides are always used in the first instance. If there is failure to control infection within 48 hours, or if the case is exceptionally severe, chloromycetin is added. In the presence of known Salmonella or Shigella infections chloromycetin is the drug of choice.

These drugs act locally on the intestinal contents and also have the advantage of being absorbed to deal with possible parenteral infections. This is important in infants where detection of parenteral infection is often difficult. It is for this reason that non-absorbable sulphonamides are not recommended.

Appropriate therapy for other complications which may aggravate the gastro-enteritis should be given when indicated. Non-specific 'remedies' such as kaolin, pectin and bismuth are not advocated. There is no good evidence that they are effective; they were used for many years without appreciable effect on the death rate. There is the possibility that such hygroscopic substances may actually accentuate water and salt loss by retaining in the bowel what might have been absorbed from it. The same may be said even more forcefully about the administration of castor oil, magnesium sulphate or other purgatives. The use of these may produce aggravation of the already excessive loss of fluid and electrolyte from the gut. In large doses they are the antithesis of the rehydration treatment of diarrhoea, which is generally recognized as one of the most striking advances in modern medicine. In homeopathic doses castor oil or magnesium sulphate are more likely to provoke or aggravate vomiting than to produce any benefit.

The principles outlined above can be applied to the treatment of diarrhoeal disorders of older children and adults. It is, however, only rarely necessary to give parenteral fluids to these groups.

Preventive measures have not been discussed here. It is obvious that the incidence of gastro-enteritis will not fall until these are more generally adopted. They include better housing, higher standards of sanitation and personal cleanliness and, above all, better nutrition.

SUMMARY

In view of the high mortality from gastro-enteritis in South Africa more efficient out-patient and home treatment is urgently needed. A scheme that has proved effective is outlined. Emphasis is placed on the value of oral and subcutaneous administration of electrolyte solutions. The role of antibiotics and other drugs is discussed briefly.

I am indebted to Prof. F. J. Ford for constant stimulus and assistance in preparation of this paper. Also to Dr. N. H. G. Cloete, Superintendent of Groote Schuur Hospital, for facilities and permission to publish. Thanks are due to Mr. B. Todt for the photographs.

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THE ASSESSMENT OF VESICAL FUNCTION IN THE PARAPLEGIC*

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Before discussing the assessment of vesical function in cord bladders, I should like to congratulate the committee responsible for the choice of a subject of such diverse medical interest. The problem is a confusing one and many questions remain unanswered. It may emphasize the urgent necessity for the establishment of several paraplegic centres in South Africa.

There will be no attempt on my part to answer the various questions or fill in the gaps. Rather shall I confine myself to a description of the experience we have gained in the paraplegic unit at Baragwanath Hospital (Fig. 1). This unit was formed in 1954 because of the incessant agitation arising as a result of the difficulty of supervision and variety of methods of treatment of paraplegics scattered throughout the wards of this large hospital. A large ward was later set aside for this purpose and to it all the paraplegics were moved, with their bed sores, contractures and indwelling catheters.

From the outset it became obvious that team work was essential. All branches of medicine and surgery were represented and took an active part in the development and management of the unit. Needless to say, the brunt of the work now fell on the shoulders of the nursing staff. Two-hourly turning of the patients was imperative in order to get the bed sores to heal, which was the most urgent aspect of all. Nutritional supervision and physiotherapy produced their desired effects and soon many of our paraplegics were in wheel-chairs or walking with the aid of crutches or calipers. Paraplegics in similar debilitated state were admitted from outlying areas, usually encumbered with a suprapubic tube and its attendant disabling features of infection and contraction of the bladder.

The prolonged and arduous techniques employed in rehabilitating these chronic cases provided a striking contrast to the comparative ease with which recent or acute paraplegics

*A paper read at the South African Urological Congress, Port Elizabeth, November 1956.

TABLE I. BLADDER CONTROL IN 90 PARAPLEGICS TREATED SINCE 1954

Site	No.	Type	No.	Bladder Control: Normal	Good	Poor	Supra-pubic
African							
Cervical	17	Complete	3	—	1	1	1
		Incomplete	14	11	3	—	—
Thoracic	48	Complete	22	—	13	7	2
		Incomplete	26	20	4	2	—
Lumbar and Cauda equina	14	Complete	2	—	2	—	—
		Incomplete	12	9	3	—	—
European							
Cervical	1	Incomplete	1	—	—	1	—
Thoracic	2	Complete	1	—	1	—	—
		Incomplete	1	—	1	—	—
Lumbar	7	Complete	1*				
		Incomplete	6	—	6	—	—
Disseminated Sclerosis	1	—	1	—	1	—	—
Total	90	Complete	29	0	17	8	3
		Incomplete	60	40	17	3	—
		Diss. Scler.	1	—	1	—	—

* Died; spinal tumour with metastases.

responded. This was particularly evident in respect of their urinary disability. By virtue of their early admission to the Unit, most of the unnecessary complications such as bed-sores and infection and contraction of the bladder were avoided and the period of rehabilitation was accordingly markedly reduced.

'If there were enough such centres, the immediate mortality could be reduced to negligible proportions and rehabilitation would be more generally effective that it is now.'[1]

Although this symposium is concerned mainly with the urinary aspects of the paraplegic, it is impossible to discuss the problem without some reference to the paraplegic as a whole. The physical and mental infirmities imparted to these patients in greater or lesser degree have a marked bearing on their future abilitites and potentialities. Conservation of every available intact neuron and muscle cell is important. The earlier this is accomplished the more rewarding is the ultimate prognosis. It is therefore necessary to stress the fact that, in the interpretation of the potentialities and capabilities of the cord bladder, the pre-existing management and treatment of the patient as a whole is intimately associated with the facility with which the functions of the bladder and its sphincteric mechanisms may be assessed.

In the actual assessment of the vesical potential the various components of the act of micturition require consideration and investigation. Normally this would entail the investigation of the detrusor muscle, the internal and external sphincters and the urethra. In the paraplegic, however, there is partial or complete loss of cerebral direction and additional forces or mechanisms such as abdominal straining or manual expression are brought into play in order to effect the emptying of the bladder. Cosbie Ross and Damanski[2] have indicated that 'the problem is to determine the ratio of the expulsive forces, that is, detrusor tone plus abdominal straining, plus manual compression, to the resistance at the bladder neck or in the urethra'.

ASSESSMENT OF THE SPECIFIC FACTORS RELATING TO VESICAL FUNCTION

The necessity for rigorous urinary supervision and control during the acute phase of spinal shock have been stressed by the previous speaker (Mr. J. A. Currie). As this stage of atonicity of the detrusor passes off, reflex activity of the detrusor returns and the behaviour of the bladder and its sphincters requires periodic survey and assessment as a guide to the future management of this vital organ. Amongst the many methods of investigation the following have proved useful:

1. *Cystometry*

This may be effected by the method pioneered by Munro,[3] which does not require expensive apparatus. The hydrostatic pressure in the bladder is measured in relation to the volume of fluid entering the bladder either continuously or by increments of 25 or 50 ml.

Although the principle is the same, more precise information may be obtained by the use of recording apparatus such as the Lewis recording cystometer.

The graphs obtained by these measurements provide an index of the activity of the bladder (Fig. 1). A flat curve indicates a flaccid atonic organ, whereas a spastic bladder is indicated by a steep rise in the pressure gradient. Detrusor activity or tone is indicated by the extent of the rise and by the presence or absence of short or sustained contraction-waves. In addition, the bladder capacity may be readily ascertained. These graphs may be adversely influenced by the presence of infection, calculi or skeletal-muscle spasm. Alone, cystometry does not give a complete picture and it should be supplemented by cysto-urethrography.

2. *Cysto-urethrography*

Initially cystometry was combined with cysto-urethrography. Sodium iodide (12½%), autoclaved in vacolitre flasks, was run into the bladder *via* the urethral catheter, or suprapubic tube, with the patient under the X-ray screen. At the same time the recording cystometer, placed in the circuit, recorded the pressure variations. As the bladder filled, its behaviour was observed under the screen and confirmatory radiographs were taken at increments of 50 ml. By this means it was hoped to obtain a visual record of the behaviour of the bladder linked with its pressure gradient. Eventually, however, the sodium iodide eroded the recording tambour and the investigations are now done separately, using normal saline for the cystometry.

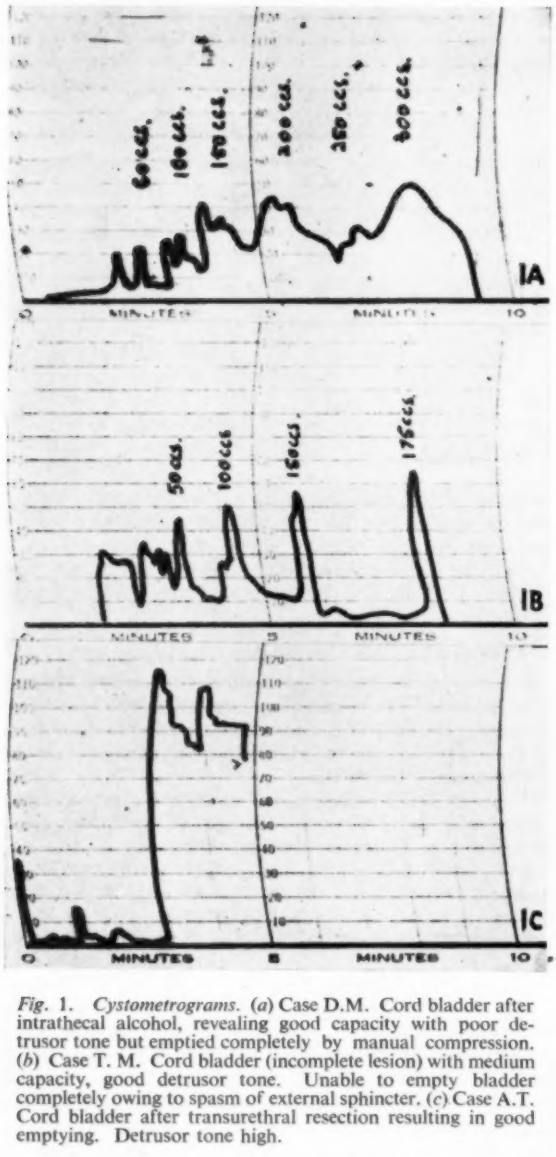

Fig. 1. *Cystometrograms.* (*a*) Case D.M. Cord bladder after intrathecal alcohol, revealing good capacity with poor detrusor tone but emptied completely by manual compression. (*b*) Case T. M. Cord bladder (incomplete lesion) with medium capacity, good detrusor tone. Unable to empty bladder completely owing to spasm of external sphincter. (*c*) Case A.T. Cord bladder after transurethral resection resulting in good emptying. Detrusor tone high.

Urethrography in the oblique position is performed before the catheter is inserted, in order to obtain information concerning the urethra and the state of the external and internal sphincters. Special apparatus is available,[6] but we have used a urethral bulb syringe with which to inject the contrast medium (Diodone 35% or similar preparation). The degree of spasm may be roughly assessed by the amount of force necessary to inject the fluid into the bladder. Caution should be observed in this respect as trauma may occur if excessive force is used. Appropriate radiographs are taken. Cystography is then proceeded with, as described above, until it becomes evident that full bladder-capacity has been reached. This is manifested by the patient's sensation of fullness, pain or burning, skeletal-muscle spasm or leakage from the urethra. The sensations experienced by the patient should be recorded for future reference. When full capacity has been reached the urethral catheter is removed or the suprapubic tube closed. If voiding occurs it is immediately visualized on the screen and radiographs are taken during the act. This is of great importance because it provides essential information on the state of the bladder neck (internal sphincter), posterior urethra and external sphincter.

If voiding does not occur or is unsatisfactory, further emptying is attempted by abdominal straining or by manual compression by the patient. The latter methods are obviously dependent on the level of the lesion and its degree—partial or complete. If the bladder cannot be emptied, further compression is attempted by the observer. At the end of the procedure the urethral catheter is then re-inserted to remove any residual sodium iodide left in the bladder. This quantity is measured, and also the amounts voided or expressed. The prime object of this important investigation is to evaluate the ability of the expulsive forces to empty the bladder. Additionally, it provides evidence of obstruction (spasm or stenosis or shelf formation) at the bladder outlet or urethra. Ureteric reflux, vesical calculi or diverticula will also be visualized. (Figs. 2 and 3.)

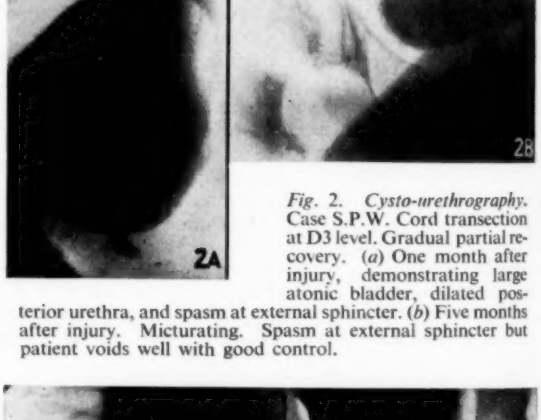

Fig. 2. *Cysto-urethrography.* Case S.P.W. Cord transection at D3 level. Gradual partial recovery. (*a*) One month after injury, demonstrating large atonic bladder, dilated posterior urethra, and spasm at external sphincter. (*b*) Five months after injury. Micturating. Spasm at external sphincter but patient voids well with good control.

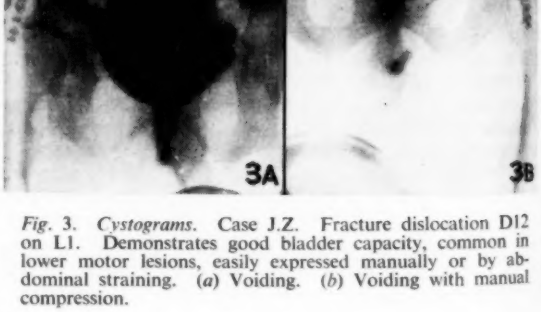

Fig. 3. *Cystograms.* Case J.Z. Fracture dislocation D12 on L1. Demonstrates good bladder capacity, common in lower motor lesions, easily expressed manually or by abdominal straining. (*a*) Voiding. (*b*) Voiding with manual compression.

It may not always be necessary to carry out time-consuming investigations requiring special apparatus. A few simple measures may provide much information of the progress being made. When a catheter is about to be changed it is of use to fill the bladder, noting its capacity and the sensations of filling, if any; and then, on removing the catheter, to observe whether natural voiding or voiding by abdominal straining or manual expression occurs. On re-inserting the catheter the residual fluid is measured. By comparison of the bladder capacity, the amounts voided or expressed and the quantity of residual urine remaining, one can determine whether and to what extent reflex detrusor activity is returning. If there is evidence of detrusor activity the catheter may then be removed and bladder training continued.[4, 5] This is done by the clock and may entail additional abdominal straining or manual compression at intervals of 1-2 hours or even longer. The ability to maintain continence for 1-2 hours or more indicates good progress. Minor degrees of incontinence during the stress of exercise should not occasion despondency.

The residual urine should be measured periodically after voiding as an index of the progress being made. Any increase in the residual urine requires full investigation as outlined above, in order to discover the cause.

3. *Panendoscopy and Cystoscopy*

These are important investigations since they afford evidence of the state of the bladder, the internal sphincter or bladder neck, the posterior urethra and possibly of the external sphincter.

4. *Sphincterometry*

This investigation is designed to complement information on the degree of spasticity or laxity at the bladder outlet. Band[6] reports encouragingly on this procedure, which entails the passage of a catheter connected to a pressure gauge in order to measure the degree of resistance at the bladder neck and external sphincter while the urethra is filled with radio-opaque dye.

5. *Intravenous or Retrograde Pyelography*

It is important to assess the functional state of the upper urinary tract periodically, since infection, calculi, hydronephrosis and hydroureter may, by their presence, have an adverse effect on the progress of the patient. An excellent survey of the upper urinary tract in paraplegia is provided by Damanski and Gibbon[7].

6. *Urinary Infection*

Prevention is better than cure is an adage which cannot be too rigidly applied in dealing with this problem in the paraplegic. Strict asepsis, the frequent changing of small catheters or polythene tubes, large fluid intake and many other factors are of fundamental importance. Periodic urinary culture of the urine and the use of appropriate antibiotics is essential. Under this regime rigorous control is required from the first moment that the paraplegic is encountered.

FACTORS RELATIVE TO THE PATIENT AS A WHOLE

1. *Nutrition*

The assessment of vesical potential cannot be complete without reference to the general state of health of the patient, for the two are closely related. A state of good physical equilibrium in the patient generally ensures easier bladder management. By comparison, a patient in a state of hypoproteinaemia, anaemia, with bed-sores, deficient vitamins, fluid and electrolytes will require correction of these deficiencies before assessment becomes of any real value. Conversely a well-balanced patient with good bladder-control may discover that the latter becomes uncontrollable if any of the deficiencies or bed-sores reappear.

2. *The Level of the Lesion*

My impression is that confusion in this respect has arisen as the result of attemtpts to associate a specific type of cord

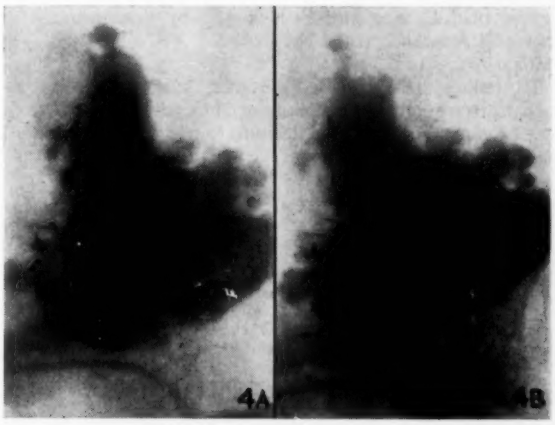

Fig. 4 (*a*) *and* (*b*). Cystogram shows contracted, pyriform bladder, with multiple diverticula, and undoubted infection arising as a result of improper management. This result may be expected if treatment is incorrect, no matter what the level of the lesion may be.

bladder with a particular level of cord injury. Cord lesions are distinct from cauda-equina lesions, because in the latter a lower-motor-neurone type of lesion exists. All bladders, however, require investigation and correct management. Failure in this respect will result in an infected, pyriform, contracted, irritable bladder with multiple diverticula no matter what the level of the lesion may be (Fig. 4).

In assessing the patient's bladder function and prognosis, his neurological status, progress and prognosis must also be considered. Signs of continuing improvement in other functions, such as motor power or sensation, indicate that bladder function may also be expected to improve *pari passu*. It is therefore wise to defer any irreversible surgical procedures on the urinary tract until one is quite certain that no further improvement will result from conservative methods. Observation of this dictum is particularly necessary in the partial lesions.

Excessive reflex muscular activity below the level of the lesion, manifested in the form of spasms, may adversely affect the controllability of the bladder. It may be necessary to employ measures such as intrathecal alcohol injection or tenotomy for the reduction of severe spasms before a true evaluation of the vesical behaviour becomes possible.

It must be remembered that a definite change occurs in the bladder and its sphincters as the stage of spinal shock

fades. The atonic state of the bladder gradually gives way to returning detrusor activity. This may vary in intensity according to the various features and factors outlined above. It is therefore mandatory to repeat the various investigations periodically in order to gain essential information and to forestall any retarding elements. For the bladder in particular, the information obtained on the state of its detrusor activity and the bladder outlet is fundamental, since without this one is at a loss to determine future procedure.

'If the expulsive forces are efficient and in spite of this the patient has a high residual urine, then there must be either a mechanical or spastic obstruction at the bladder neck or in the urethra. As a corollary to this, it is obvious that, even if the expulsive forces are weak, satisfactory evacuation of the bladder may still be achieved by diminishing the degree of resistance at the bladder neck'. (Cosbie Ross and Dananski[2]).

To reduce bladder-neck resistance, surgical procedures such as transurethral resection or pudendal nerve section may be carried out; but the decision to use these procedures must be made by application of the above investigations.

SUMMARY

1. The method of assessment of vesical function and its sphincteric mechanisms is briefly discussed.

2. The results obtained by such periodic assessment are of great assistance in the management and treatment of the urological problems of the paraplegic.

3. A plea is made for early correct management of the paraplegic, preferably in spinal centres. Effective early treatment diminishes the complications which may arise and reduces greatly the time required for rehabilitation.

4. A further plea is made for the establishment of several spinal centres throughout the country.

Thanks are due to Dr. J. D. Allen, Superintendent, Baragwanath Hospital, for the development of the Paraplegic Unit. Mr. S. Kleinot, Senior Surgeon, is thanked for constant encouragement and invaluable assistance; the Radiology Department and Drs. M. Brukin and Mr. G. Schepers for their aid in cysto-urethrography; Mr. R. Lipschitz and Mr. J. Block, of the neurosurgical team, for their advice; Mr. E. A. Wilkinson and Dr. S. Kimmel for photographic assistance, and Mr. Haywood of the Records Department, Baragwanath Hospital, for constant help.

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7. Damanski, M. and Gibbon, N. (1956): *Ibid.*, **28**, 24.

ASSOCIATION NEWS : VERENIGINGSNUUS

SOUTHERN TRANSVAAL BRANCH, ANNUAL MEETING

In his report for the year 1956-57 the Hon. Secretary, Dr. W. Girdwood, stated that the membership at the end of December 1956 numbered 1,412, an increase of 40 over 1956. The deaths of 11 members were recorded, viz. Drs. J. D. Allen, P. J. Booysen, Reuben Burns, L. J. Coetzee, A. E. Dreosti, Felix du Toit, H. Haden, R. C. J. Meyer, Margaret Orford, L. S. Williams and M. A. Shapiro.

At clinical meetings, at which the attendance varied from 50 to 75, addresses were given by Prof. Sir Robert MacIntosh of Oxford, Prof. C. H. G. Macafee of Belfast, Dr. Robert M. Stecher of Cleveland, Ohio, and Drs. B. M. Bloomberg and P. Menhof of Johannesburg. At an address by Dr. Sheila Sherlock of London 400 attended. At a seventh clinical meeting 4 cases were demonstrated and discussed. Seven business meetings of the Branch were held, at which the attendances were 105, 95, 70, 64, 40 (2) and 26.

The work undertaken by the Branch Council and Committees in 1956 greatly exceeded that done in any previous year. Branch Council meetings commonly lasted 3½-4 hours and were followed by another 4 hours at an adjourned sitting. During the year, 189 meetings were held. Much attention was given to liaison with the Press, and through the magnificent effort of Dr. Hillel Shapiro a Branch Newsletter was published. Greatly increased negotiations had resulted from Federal Council's resolution declaring that future appointments to benefit societies should be on the basis of open panels. Much attention was devoted to the schedule of fees for medical aid societies. A disciplinary system has been in use to prevent over-visiting and to limit the societies' expenditure per patient. Dr. Girdwood referred in his report to the impotence of the rule of the Medical and Dental Council concerning the acceptance of appointments under conditions 'derogatory to the medical profession', and to the sense of futility that resulted.

The Telephone Sub-committee had met fortnightly and had dealt with 300-400 applications a month.

Over a series of years the Southern Transvaal Branch had raised over £3,000 for the Benevolent Fund through its Annual balls. From the 1956 annual ball £583 was remitted. The annual dinner was held at the Skyline Hotel, Johannesburg; 75 members were present.

Two Divisions of the Branch have come to an end, viz. the Vereeniging Division, which becomes a Branch, and the S. E. Transvaal and Swaziland Division, which has ceased.

Report of Hon. Treasurer, Mr. J. Wolfowitz

Mr. Wolfowitz in presenting the audited Branch accounts reported a deficit (excess of expenditure over income) of £214, notwithstanding a grant of £500 received from Federal Council, and a nett increase of £242 income from subscriptions. He urged that the finances of the Branch were in a serious state and called for serious and urgent attention.

The Branch's property at Medical House, Esselen Street, Johannesburg, was in urgent need of repairs, and these were being proceeded with at an estimated cost of £1,250.

NORTHERN TRANSVAAL BRANCH, ANNUAL MEETING

The Annual General Meeting of the Northern Transvaal Branch was held at the General Hospital, Pretoria, on 12 February 1957, the President, Dr. B. Epstein, taking the chair.

The following office-bearers were elected for the ensuing year: *President*, Dr. F. Ziady; *Vice-president*, Dr. J. G. du Toit; *Hon. Secretary*, Dr. E. Fasser; *Hon. Treasurer*, Dr. F. W. McLachlan; *elected members of Branch Council*—Drs. W. H. Lawrance, J. H. Casewell and C. M. Grundlingh.

The following representatives of the Pretoria Division were also elected to Branch Council, viz. Drs. S. Donen, D. A. Fowler, G. v. R. Mostert, W. E. B. Lawrance, J. van Duyn, P. N. Swanepoel, G. K. Loveday, L. Klein, T. H. R. Bohlmann, C. L. Laubscher, J. F. P. Erasmus, O. E. Hansen, A. Glen, F. A. K. van Wyk and N. L. Murray, and Profs. F. G. Geldenhuys, J. G. A. Davel and C. H. Derksen.

Dr. E. Fasser, in presenting his secretarial report, stated that the membership of the Branch was 551 (Divisions: Pretoria 369, Pietersburg 70, Eastern 75, Western 37)—an increase of 39 over 1956. The clinical material offered at Branch meetings had been of ever increasing quality, invaluable to busy practitioners with little time to read; but it appeared that most members were too busy to avail themselves of the advantage offered. The speakers included Sir Robert McIntosh, Mr. A. Miller and Mr. E. A. Nicol (from the UK), and Prof. J. H. Louw (from Cape

Town); and a surgical symposium was arranged by the Pretoria University.

Dr. Fasser went on to comment on the lack of interest in Association affairs displayed by members, and mentioned in this connection the unrest and dissatisfaction around us, involving public and practitioner alike, the over-production of doctors and the inability of the Association to apply disciplinary action to its members. The Branch Council, working locally and through its Federal Council representatives was endeavouring to remedy the situations as far as possible.

The Branch Council had met monthly and the meetings were often protracted. Much confusion had been caused by the sudden and unexpected directive from Federal Council that all sick funds should be induced to operate on the open-panel system, and only now was this matter being cleared up. Dr. Struthers, the retiring President of the Association, had been a pillar of strength on the Branch Council and its committees as well as on Federal Council committees.

The Hon. Secretary referred to the short 'apprenticeship' which had been arranged with the University of Pretoria for final-year medical students to serve with general practitioners in Pretoria (on a voluntary basis). Some of the doctors reported that they had found the relationship to stimulate academic interest on their part. The University had also held refresher courses for medical practitioners. The Branch had donated to the Pretoria University's Merensky Library the historical library which had been collected under the guidance of Dr. L. Klein and with the advice of Dr. Guthrie of Edinburgh. The Branch continued to make its grant of £100 a year to the Pretoria University Library.

A successful dinner-dance had been held at the Pretoria Country Club.

Dr. E. A. Donegan, Hon. Treasurer, submitted the audited accounts for 1956. The excess of income over expenditure was £481, and the accumulated Branch funds now stood at £3,531.

The outgoing President, Dr. B. Epstein, after inducting Dr. F. Ziady as President, delivered his valedictory address, *The Selection of Medical Students*.[1]

1. Epstein, B. (1957): S. Afr. Med. J., **31**, 291.

NATAL COASTAL BRANCH, ANNUAL MEETING

The annual general meeting of the Natal Coastal Branch was held in Durban on 13 February 1957 (President, Mr. Lance Knox). The following office-bearers were elected: *President*, Dr. N. A. Rossiter; *Vice-President*, Dr. N. R. Pooler; *Hon. Treasurer*, Dr. F. Walt; *Hon. Secretary*, Dr. K. W. Dyer; *Associate Secretary*, Dr. David Martyn; *elected members* of Council—Drs. S. Disler, R. Mundy and J. Stolp. The *ex officio* members of Council are Mr. Lance Knox (Past-president), and the following Federal Council members: Dr. A. Broomberg, Dr. E. W. S. Deale, Dr. H. Grant Whyte, Dr. N. A. Rossiter and Dr. Alan B. Taylor.

Dr. K. W. Dyer, in presenting the annual secretarial report stated that the Branch membership exceeded 500. The following members had died during the year: Drs. Sidney Ernest Aubrey, Michael Gilbey, George Herbert Gunn, and Cecil Hobart Houghton.

Under the chairmanship of Dr. A. Broomberg much progress was made by the Medical Congress Committee with the organization of the Congress to be held in Durban in 1957.

A successful and well-attended annual dinner was held in October 1956 at the Marine Hotel, Durban, when the Minister of External Affairs, Mr. Eric Louw, was the guest of honour and delivered an address, which was much appreciated, on the subject of the individual citizen—the patient—as a productive unit and a matter of concern to the family, the community and the State.

Dr. Aubrey Beiles, Hon. Secretary of the Clinical Sub-committee (Chairman, Dr. Pauline Klenerman) reported that during 1956 10 clinical meetings had been held, 3 of which were clinical discussions at the Addington, King Edward VIII and King George V Hospitals, and 4 were lectures by prominent visitors from overseas. The regular monthly meetings held by the following were open to Branch members: The Departments of Medicine and of Pathology of the Medical School, the King George V and Addington Hospitals, and the Radiologists of the King Edward VIII Hospital.

NATAL INLAND BRANCH, ANNUAL MEETING

The annual general meeting of the Natal Inland Branch was held at Grey's Hospital, Pietermaritzburg, on 8 February 1956 at 8 p.m. The President, Dr. H. A. Kalley, took the chair and 28 members were present.

In his annual report the President stated that the membership of the Branch at the end of 1956 stood at 190. During the year Dr. R. D. Kidd had been elected as an Emeritus Member of the Association. Three visiting lecturers (Mr. B. W. F. Bishop, Mr. I. Barnat and Dr. A. Janovics) had addressed meetings of the Branch and several clinical meetings as well as numerous business meetings had been held.

The following office-bearers were elected: *President*, Dr. N. Mungo Thompson; *President-elect*, Dr. R. B. Peckham; *Vice-president*, Dr. R. J. Reznek; *Hon. Secretary*, Dr. T. H. Whitsitt; *Hon. Treasurer*, Dr. R. P. Seymour; *Clinical Secretary*, Dr. D. Lithgow; additional members of Branch Council—Drs. L. R. Tibbit, C. Akerman and C. A. Ovendale.

Dr. Mungo Thompson was welcomed as President by Dr. Kalley, who then vacated the chair.

The regulations for the training of midwives were referred to the Obstetrical and Gynaecological Group with a view to consideration by Federal Council.

The meeting expressed the opinion that composite fees for team-work in cardiac and brain surgery were not unethical. It was also resolved that Ethical Rule 16 of the Medical and Dental Council (professional secrecy) was vague and ambiguous and should be clarified and amplified.

QUESTIONS ANSWERED

CONTROL OE THERAPEUTIC SUBSTANCES

Question 1

Are there any regulations in this country which govern the manufacture of intravenous preparations?

Answer: The quality of all drugs sold in South Africa is governed by the Foods, Drugs and Disinfectants Act No. 13 of 1929. In addition certain listed drugs are controlled, as regards strength, quality and purity, by the Therapeutic Substances Regulations framed under the Medical, Dental and Pharmacy Act No. 13 of 1928.

The only intravenous preparations controlled by the latter regulations are preparations of human blood and plasma substitutes as defined in the schedule, and intravenous preparations of antibiotics and vitamins, whereas other intravenous preparations, e.g. normal saline, dextrose solutions, etc., are controlled only by the former Act.

Question 2

Is the manufacturer of such preparations obliged to test by chemical analysis the purity of the ingredients used by him? In other words is he permitted to use a substance sold to him as sodium chloride without satisfying himself by chemical analysis that he has purchased the correct article?

Answer: Medicinal preparations falling under the Foods, Drugs and Disinfectants Act must comply with the standards prescribed by the British Pharmacopoeia or any addenda thereto and, although the Act does not specify how the manufacturer is to comply with these standards, the onus is on him to do so.

Scheduled therapeutic substances manufactured in or imported into the Union of South Africa must comply with the standards for strength, quality and purity as prescribed in the relevant schedules to the regulations, which also prescribe the tests for

ensuring that each batch of a preparation complies with these standards.

Question 3

Is there any regulation which states that the finished product must be subjected to biological tests to determine whether it is safe?

Answer: The standards as prescribed by the British Pharmacopoeia or addenda thereto may include biological standards and the manufacturer is required to comply with these standards.

The Therapeutic Substances Regulations lay down definite biological tests that must be carried out on all batches of intravenous preparations falling under them before they may be released for sale, and the manufacturer must keep full protocols of these tests to the satisfaction of the licensing authority. Batches of such preparations may not be released for sale unless they have passed these tests.

Question 4

Are intravenous preparations manufactured on licensed premises which are built to certain standards?

Answer: Therapeutic substances may only be manufactured in or imported into the Union by persons licensed so to do by the licensing authority, which is the Minister for Health. The licensee must provide and maintain adequate staff, premises and plant for manufacturing and testing of his products to the satisfaction of the licensing authority and must maintain proper records. His premises and records are open to inspection by duly authorized inspectors. Manufacturers of intravenous preparations not falling under the Therapeutic Substances Regulations have their premises inspected by the Department of Labour and they are also subject to inspections by duly authorized inspectors under the Food, Drugs and Disinfectants Act. Thus premises do not have to be licensed but are subject to inspections to ascertain if they are adequate for the type of manufacture to be undertaken.

These answers, by Dr. J. E. C. Mullen, Pharmacologist, Union Health Department, are published with the permission of the Secretary for Health.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Notensil—New major tranquillizer

Benger Laboratories announce that their Associate Company. Fisons Chemicals (S.A.) (Pty) Limited, are marketing a new major tranquillizing agent, acetylpromazine, named Notensil, and supply the following information:

Pharmacological studies have proved acetylpromazine to be twice as potent weight for weight as the well-known chlorpromazine hydrochloride. Moreover, extensive clinical appraisal of the product both in the United Kingdom and France has shown Notensil to be less toxic than any other major tranquillizer. In particular, no jaundice or agranulocytosis, and no skin sensitivity, have been observed.

Indicated primarily for the treatment and control of certain psychoses (e.g. mania, schizophrenia, confusional states), in some anxiety states, and for alcoholism and drug addiction, Notensil will also be of value for the relief of early-morning vomiting in pregnancy, the relief of vomiting due to drugs, infections etc. and, in association with analgesics, for the relief of intractable pain. Dosage is variable but a total daily dosage of 150-200 mg. is envisaged for major psychoses, while a single dose of 10-30 mg. will be found to be suitable in the other conditions named.

Notensil is available in two tablet strengths: 10 mg.—in bottles of 30 and 1,000; and 25 mg.—in bottles of 100 and 1,000. A parenteral form will be available shortly.

Mysteclin Suspension

Mysteclin Suspension a complementary product to Mysteclin Capsules is now available for the treatment of many common infections which respond to tetracycline therapy. The following information is supplied by the manufacturers.

Mysteclin Suspension for oral use is a ready-to-take, fruit-flavoured, corn-oil suspension containing two important antibiotics viz, Steclin (Squibb Tetracycline) for broad-spectrum antibacterial therapy, and Mycostatin (Squibb Nystatin) for effective antifungal prophylaxis. Each 5-c.c. teaspoonful of the suspension contains the equivalent of 125 mg. of Steclin hydrochloride and 125,000 units of Mycostatin.

It is available in 60-c.c. (2-ounce) bottles. The preparation is stable for 18 months.

Action. Steclin provides antimicrobial action similar to that of other broad-spectrum antibiotics. Mycostatin the first safe antifungal antibiotic, has pronounced action against *Candida Monilia albicans*.

Use. The combined administration of Mycostatin and Steclin as provided by Mysteclin, affords both antimicrobial therapy with a broad-spectrum antibitotic as well as safe and effective prevention of fungal superinfection which may occur as a result of therapy with broad-spectrum antibiotics, especially when such therapy must be intensive or prolonged. This preparation is now available in a dosage form particularly suited to pediatric practice.

Dosage. For infants and children the usual dosage should supply approximately 20 mg. of tetracycline per kg. of body weight each day in divided doses. The minimum dosage for adults is 1 g. of tetracycline each day in divided doses (e.g. 2 teaspoonsful of Mysteclin Suspension 4 times daily).

Squibb Laboratories (Pty) Limited, Pharmacy House, Jorissen Street, Braamfontein, Johannesburg. P.O. Box 9975. Telephone 44-9648.

Proladone, Long-acting Analgesic

Chemistry. Proladone is dihydrohydroxycodeinone pectinate, a slightly viscous aqueous solution, introduced by The Crookes Laboratories Ltd.

Pharmacology. The urinary excretion of dihydrohydroxycodeinone after injections of Proladone and dihydrone hydrochloride has been studied in human volunteers. The alkaloid appeared later in the urine and was present for twice as long following Proladone as following dyhydrone hydrochloride. Half the alkaloid was excreted within 1 hour following dihydrone hydrochloride and within 2½ hours following Proladone; 86·6% of the alkaloid injected was recovered in the case of Proladone and 65% in the case of dihydrone hydrochloride. These findings confirm the prolonged action of Proladone and provide evidence that the *pectinate* is more resistant to destruction in the body than the hydrochloride.

Post-operative Pain Control. In a series of 18 patients Belam (1956) found that with one injection (10 mg.) of Proladone given before the operation, no patient required any post-operative sedation within the next 14 hours. Only one patient in this series required any post-operative sedation at all. These findings were confirmed in a further series of 100 cases. Probably it is fair to say that, on an average, the effects of Proladone last 3 times as long as those of morphine. Mandel (1954) reported 2 or 3 successive nights free from pain and insomnia resulted from single injections of Proladone (10 mg.) given in the evening to patients who had undergone orthopaedic operations. In these patients, morphine produced only short-lived and unsatisfactory analgesia.

Control of Pain due to Inoperable Carcinoma. Both Mandel and Belam found that Proladone produced satisfactory pain relief in cases of inoperable carcinoma.

Side-effects. Unwanted side effects are fortunately few. There appears to be only a slight depressant effect on respiration and a relatively low incidence of nausea and vomiting. Proladone has no effect on renal function.

Administration and Dosages: Intramuscular injection—it should

not be given intravenously. The usual dose is 1 ml. (10 mg. of dihydrohydroxycodeinone) and this can be repeated when required.

Packings: 1 ml. ampoules in boxes of 6.

Sole South African Agents, Messrs. South African Druggists (Division Sive Bros. & Karnovsky), Jeppe Street, Johannesburg. Further details can be obtained from The Crookes Laboratories Ltd., P.O. Box 1573, Johannesburg.

'*Acalo*' (*Phenaglycodol, Lilly*).

Messrs. Eli Lilly's new product 'Acalo', which was announced in the last issue of the *Journal*[1] was originally introduced under the trade name 'Ultran' (Phenaglycodol, Lilly) in the United States of America.

1. New Preparations and Appliances (1957): S. Afr. Med. J., **31**, 437.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. Jack Penn, F.R.C.S. (Edin.), has been invited by an American organization for the rehabilitation of Japanese atom-bomb casualties to take a plastic-surgery team to Hiroshima with a view to carrying out treatment in plastic surgery on the spot, and at the same time to assess future plastic surgical requirements.

Mr. Penn will be leaving on 12 May for the USA to plan the details of the project, to meet the rest of the personnel, and to assemble his equipment.

* * *

Radiological Society of South Africa. The next monthly meeting of the Transvaal Sub-group of the Radiological Society of South Africa will take the form of a symposium on *The Radiation Hazards of Diagnostic X-rays*. Chairman, Dr. A. Agranat. Speakers, Dr. M. H. Fainsinger, Dr. D. Durbach, and Dr. P. V. Tobias. The meeting will be held in the Harveian Lecture Theatre of the Medical School, Johannesburg, on Tuesday, 28 May 1957, at 8.15 p.m. All medical practitioners are invited to attend.

The Transvaal Sub-group draws attention to the following statement issued by the United Nations Scientific Committee on the Effects of Atomic Radiation:

'Information received so far indicates that, in certain countries, by far the most important artificial source of irradiation is the use of radiological methods of diagnosis and that this may be equal in importance to radiation from all natural sources. It is possible that such radiation may be having a significant genetic effect on the population as a whole. The United Nations Committee is fully aware of the importance and value of the medical use of radiations but wishes to draw the attention of the medical profession to these facts and to the need for a more accurate estimate of the amount of exposure from this source.'

Readers of the *Journal* are referred to the abov ementioned statement of the United Nations Scientific Committee,[1] which was published in the *Journal* of 2 March 1957 at page 203, and to Dr. D. Durbach's review[2] of the subject of Radiation Hazards at page 187 of the same issue.

1. U.N. Scientific Committee (1957): S. Afr. Med. J., **31**, 203.
2. Durbach, D. (1957): *Ibid.*, **31**, 187.

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Congresses in Anaesthetics. The Australian Society of Anaesthetists, in conjunction with the German and Swiss Societies of Anaesthetists, will be holding a Congress in Vienna on 13-15 June 1957. The main theme will be, 'Problems of post-operative treatment'.

The French National Congress of Anaesthesiology will take place in Marseilles on 18-21 July 1957.

Further information in regard to these events may be obtained from the Hon. Secretary of the South African Society of Anaesthetists, P.O. Box 558, Pretoria.

* * *

Dr. Ivor M. Shulman, F.R.C.S. (Eng.), D.M.R.T. (Lond.), radiotherapist, of 612 Dumbarton House, Church Street, Cape Town (telephone: rooms, 2-8811, residence 69-5085), has been authorized by the Atomic Energy Board to use radio-active isotopes for all diagnostic and therapeutic purposes, and is now in possession of the necessary facilities to undertake these proceedings.

Dr. Ivor M. Shulman, F.R.C.S. (Eng.), D.M.R.T. (Lond.), radioterapeut te Dumbarton-Huis 612, Kerkstraat, Kaapstad (telefoon: spreekkamers, 2-8811, woning 69-5085) is deur die Atoomkragraad gemagtig om radio-aktiewe isotope vir alle diagnostiese en terapeutiese doeleindes te gebruik en is nou in besit van die nodige geriewe om hierdie metodes toe te pas.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van ernstige epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk 16 April—25 April 1957.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	7	4	—	—	11
Kaapprovinsie ..	17	10	7	—	34
Oranje-Vrystaat ..	6	4	—	—	10
Natal	16	11	—	—	27
Totaal ..	46	29	7	—	82

Notifications during the period 26 April-2 May 1957.

	Eur.	*Nat.*	*Col.*	*Asiat.*	*Total*
Transvaal	14	11	2	—	27
Cape Province ..	12	7	12	—	31
Orange Free State	2	1	—	—	3
Natal	7	6	—	1	14
Totals	35	25	14	1	75

Pes, Pokkies, Tifuskoors: Geen.

REVIEWS OF BOOKS : BOEKRESENSIES

OBSTETRICS AT IBADAN

Clinical Report of the Department of Obstetrics University College, Ibadan, Nigeria. April 1st, 1953-December 31st, 1954. By J. B. Lawson, M.A. (Cantab.), M.B., B.Chir., M.R.C.O.G. and U. G. Lister, M.B., B.S. (Lond.), M.R.C.O.G. Pp. 170. University College, Ibadan, Nigeria. 1956.

Contents: Foreword. Introduction. *Obstetric Report.* Statistical Summary. Critical Review. Tables: 1. Associated Diseases and Conditions not detailed elsewhere. 2. Anaemia of Pregnancy. 3. Pre-eclampsia, Essential Hypertension, and Chronic Nephritis. 4. Eclampsia. 5. Accidental Haemorrhage. 6. Placenta Praevia. 7. Persistent Occipito-posterior Position and Deep Transverse Arrest. 8. Uncomplicated Breech Delivery. 9. Complicated Breech Delivery. 10. Face and Brow Presentations. 11. Transverse and Oblique Lie (in labour). 12. Multiple Pregnancy. 13. Labour following Previous Caesarean Section. 14. Prolapse and Presentation of the Cord. 15. Post-partum Haemorrhage. 16*a*. Manual Removal of Placenta (delivered in hospital). 16*b*. Retained Placenta (B.B.A.). 17. Caesarean Section. 18. Forceps Delivery. 19. Version in Labour. 20. Embryotomy and Craniotomy. 21. Obstructed Labour. 22. Ruptured Uterus. 23. Puerperal Pyrexia (Genital tract infection). 24. Puerperal Pyrexia (Extra-genital infection). 25. Maternal Deaths. *Paediatric Report.* Statistical Summary. Critical Review. Tables: 1. Stillbirths. 2. Neonatal Deaths. 3. Premature Live-born Infants. 4. Congenital Malformations. 5. Birth Trauma.

This is the first report issued from the Department of Obstetrics of the University College, Ibadan, Nigeria, and covers the work in that department over the period of 20 months from April 1953 to December 1954. At the outset one must congratulate Drs. Lawson and Lister who compiled this monumental, yet magnificent

work. To many people in more advanced countries the material presented in this report may sound almost unbelievable, as to them those types of obstetrical difficulties are probably unhappy relics of bygone days. We in South Africa, whilst assuredly being placed in a more favourable obstetrical position than that under which these valiant people had to labour, must acknowledge that, by and large, we can fully appreciate their difficulties. This is particularly true as far as the type of obstetrical case so peculiar to the Native peoples of Africa is concerned. Where in a civilized community will one still read of 49 craniotomies listed in a report covering a period of 20 months?

Tropical diseases such as malaria, dysentery, bilharzia and profound anaemia (probably due to dietetic deficiency of protein, for iron deficiency was not prominent) are rife in Nigeria and present obstetrical complications which are seldom seen in other parts of the world. An interesting feature is the relative frequency of intrapartum and postpartum procidentia. In South Africa the relative rarity of prolapse amongst the Bantu has been the subject of many interesting conjectures. Twelve cases of vesico-vaginal fistula following obstructed labour are listed. This complication is still all too common amongst our own Bantu people. The incidence of multiple pregnancy is high (1 in every 24 pregnancies). The rates for Caesarean section and forceps delivery were low, both being 2·3% of the total deliveries. In both series, inertia was an uncommon indication. Cardiac disease and pre-eclampsia were uncommon complications of pregnancy.

One can only praise the authors on something well done.

W.H.M.

SURGICAL PATHOLOGY

A Textbook of Surgical Pathology. Seventh Edition. By Charles F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S. (Edin.) and Bruce M. Dick, M.B., F.R.C.S. (Edin.). Pp. viii + 730. 322 Illustrations. 63s. net. London: J. & A. Churchill Ltd. 1956.

Contents: Preface to Seventh Edition. Preface to First Edition. I. The Response to Injury. II. Constitutional Effects of Injury. III. Wound Infections. IV. Transplantation and Preservation of Tissues. V. Tuberculosis. VI. Actinomycosis. VII. Hydatid Disease. VIII. Tumours. IX. Diseases of Bones. X. Diseases of Joints. XI. Diseases of Muscles, Tendon Sheaths and Bursae. XII. Diseases of Blood Vessels. XIII. Diseases of Lymph Glands and Vessels. XIV. Diseases of the Skull and Brain. XV. Diseases of the Spine and Spinal Cord. XVI. Diseases of the Peripheral Nerves. XVII. Diseases of the Thorax. XVIII. Diseases of the Pericardium, Heart and Great Vessels. XIX. Diseases of the Breast. XX. Diseases of the Mouth, Jaws, Salivary Glands and Neck. XXI. Diseases of the Thyroid Gland. XXII. Diseases of the Pharynx, Larynx and Oesophagus. XXIV. Diseases of the Stomach and Duodenum. XXV. Diseases of the Small Intestine. XXVI. Diseases of the Colon. XXVII. Diseases of the Vermiform Appendix. XXVIII. Diseases of the Peritoneum. XXIX. Diseases of the Gall-bladder, Liver and Bile Ducts. XXX. Diseases of the Pancreas. XXXI. Diseases of the Spleen. XXXII. Diseases of the Adrenal Glands. XXXIII. Diseases of the Urinary Organs. XXXIV. Diseases of the Male Generative Organs. XXXV. Diseases of the Female Generative Organs. Index.

This text-book of surgical pathology has stood the test of time well. The new edition—the 7th in 24 years—will enhance its reputation. It presents virtually all the surgical pathology required for the purposes of examinations at all levels in surgery, and is also a book to which one turns readily for a balanced and authoritative opinion when seeking to read round problems of an unusual nature in practice. It is not possible in a book of this size to discuss all opinions regarding the pathogenesis and progress of even most of the problems of surgery. Exhaustiveness in a text-book tends towards exhaustion in a reader. This book is not exhaustive by that standard, but it gives a sane exposition of most of the facts of surgical pathology and avoids dallying with the fancies.

The sections dealing with surgical physiology and surgical infections have been suitably re-written in view of the changing emphasis on such matters, and as a result the systemic and special response to injury, the transplantation and preservation of tissues and the functions of radio-isotopes replace, in extent at least, the details of coccal infective processes and the vanishing disease of syphilis.

The chapters on anuria and the abnormalities of the oesophagus have also been rewritten—although the word 'short' on page 455 should be written in inverted commas.

Altogether this book is a worth-while purchase for anyone interested in surgical pathology from the point of view of general reading, or study or teaching.

R.D.H.B.

THE FATHER OF ORTHOPAEDIC SURGERY

The Life of Hugh Owen Thomas. By David le Vay, M.S., F.R.C.S. Pp. vii + 135. XIII Plates and 13 figures. 25s. net. Postage 11d. Abroad. Edinburgh: E. & S. Livingstone Ltd. 1956.

Mr. Le Vay has contributed a lively and absorbing biography of one of the great medical figures of the 19th century. Hugh Owen Thomas was the father of orthopaedic surgery as we know it. The author elaborates his picture of a surgeon who was essentially a physician and whose creed was founded on the natural principles of rest, reaction and repair, and who insisted that surgery should be used only to speed and not to impede the repair processes of nature.

In describing the development of the individualistic and anti-authoritarian character of Thomas, he gives us also an interesting vignette of his times and of his contemporaries, who included Hilton, Benjamin Brodie and James Paget. When Thomas was a medical student at Edinburgh, Lister was house surgeon to Syme. And, when we realize that Koch discovered the tubercle bacillus only 9 years before Thomas died and that X-rays were used for the first time some 5 years later, we appreciate the more the stature of the man on whose concepts the modern practice of orthopaedics is based.

The illustrations are beautifully produced and this monograph will grace the shelves of all those interested in the history of medicine in general and of orthopaedic surgery in particular.

A.J.H.

PSYCHOPATHY AND DELINQUENCY

Psychopathy and Delinquency. By William McCord, Ph.D. and Joan McCord, Ed.M. Pp. x + 230. 86·50. New York and London: Grune & Stratton, Inc. 1956

Contents: Foreword. Preface. 1. Who is the Psychopath? The Psychopath is Asocial. The Psychopath is Driven by Primitive Desires. The Psychopath is Highly Impulsive. The Psychopath is Aggressive. The Psychopath Feels Little Guilt. The Psychopath Has a Warped Capacity for Love. The Psychopathic Syndrome. Notes. 2. From Moral Insanity to Psychopathy. Notes. 3. The Problem of Diagnosis. Notes. 4. The Causes of Psychopathy. The Hereditary Approach. The Neurological Approach. The Environmental Approach. A Neuro-Social Theory of Causation. The Effects of Social and Cultural Forces. Notes. 5. The Treatment of Psychopathy. The Treatment of Adult Psychopathy. The Treatment of Child Psychopathy. Notes. 6. Milieu Therapy: an Evaluation. Aims. Methods: A. The Diagnostic Problem. B. The Measurement Problem. The Effects of Wiltwyck Treatment: A. The Findings of the 1954 Study. B. The Findings of the 1955 Study. Conclusions: A. Milieu Therapy's Effects on Neurotic and Psychotic Children. B. Milieu Therapy's Effects on Behavior Disorders. C. Milieu Therapy's Effects on Child Psychopaths. D. Summary of All Changes. E. Why do the Changes Occur? Notes. 7. The Psychopath, The Law, and Society. The Psychopath and Legal Policy. The Psychopath and Social Policy. Notes. Appendix. A. Wiltwyck's Population. B. Tests and Ratings. C. Measurement Procedure, Validity and Reliability. D. Results of 1954 Study. E. Counselor-Boy Relations and the Effect on Personality. Index.

The authors start with their definition of psychopathy. The psychopath to them is asocial (meant is antisocial), he is driven by primitive desires, he is highly impulsive, he is aggressive, he has no feelings of guilt, and he has a warped capacity of love. To many this definition would seem too narrow; by considering antisocial tendency an essential symptom of psychopathy, the authors must obviously confine themselves to the criminal or delinquent psychopaths (as in fact the title of their book indicates). They discuss the causes of psychopathy—hereditary, neurological (encephalitis and lesions of frontal lobe and hypothalamus) and environmental. It is the old question of endogenous and exogenous causation of mental diseases, in reality there is always the combination of the two. Up till recently the endogenous factors were considered the most important in psychopathy; the authors (although not denying the possibility of hereditary factors) are inclined to stress the importance of environmental factors, which is of course a more optimistic standpoint where therapy is concerned. The authors then proceed to discuss the treatment of psychopathy both in the adult and the child. Of the various forms of treatment psychotherapy (difficult and very often disappointing as it may be) seems to offer the best chances, especially the so-called milieu therapy as introduced by Aichhorn (mobilizing the child's total environment for therapeutic purposes).

These children have been brought up without affection and have suffered unreasonable severity and brutality. One has first of all to compensate for this great lack of love: 'The treatment should be characterized thus: a consistently friendly attitude, wholesome

occupation, plenty of play to prevent aggression, and repeated talks with individual members'.

Chapter 6 is an evaluation of this milieu therapy as applied in the Miltwyck School for Boys in New York. This most important chapter contains the authors' actual research work, and cannot be reviewed in a few words. It should be read, the results are very impressive and almost hopeful, certainly if compared with the completely negative attitude towards psychotherapy and the hopeless results of the past.

In the last chapter—the Psychopath, The Law and Society—the authors discuss the interesting question of responsibility for criminal actions and stress the necessity of early treatment of psychopathic children.

'If society has the will, it can protect itself against this most dangerous and, at the same time, most lonely of human beings'.

F.H.K.

GYNAECOLOGY

Shaw's Text-book of Gynaecology. Seventh Edition. Revised by John Howkins, M.D., M.S., F.R.C.S., F.R.C.O.G. Pp. vii + 704. 4 Coloured Plates and 352 Text-figures. 31s. 6d. net. London: J. & A. Churchill Ltd. 1956.

Contents: I. Anatomy. II. Normal Histology. III. Physiology. IV. Gynaecological Diagnosis. V. Malformations of the Female Generative Organs. VI. Diseases of the Vulva. VII. Diseases of the Vagina. VIII. Venereal Diseases of the Female. IX. Tuberculosis of the Genital Tract. X. Injuries of the Female Genital Tract. XI. Diseases of the Urinary System. XII. The Pathology of Conception. XIII. The Pathology of Pregnancy: Abortion. XIV. The Pathology of Pregnancy: Hydatidiform Mole and Chorion Epithelioma. XV. The Pathology of Pregnancy: Ectopic Gestation. XVI. Disorders of Menstruation. XVII. Hormone Therapy in Gynaecology. XVIII. Inflammations of the Uterus. XIX. Prolapse. XX. Displacements. XXI. New Growths of the Uterus: Connective Tissue Tumours. XXII. New Growths of the Uterus: Epithelial Tumours of the Cervix. XXIII. New Growths of the Uterus: Epithelial Tumours of the Body. XXIV. Radiotherapy in Gynaecology. XXV. Endometriosis. XXVI. Diseases of the Ovaries. XXVII. Inflammations of the Uterine Appendages. XXVIII. Diseases of the Broad Ligament, Fallopian Tubes and Parametrium. Appendix. Index.

The late Wilfred Shaw's 'Text-book of Gynaecology' has appeared in its 7th edition—ably revised by John Howkins.

This book is well conceived, is beautifully written, and contains all that can be expected from a text-book. The illustrations are clear, concise and well distributed. Mr. Howkins has a flowing easy style which will be much enjoyed by the reader.

A large section of the book has naturally been altered and every aspect has been brought up to date. The chapter on cancer of the cervix is exceptionally well done.

This book is recommended to any general practitioner wishing to read informatively in gynaecology and to all medical students. An important item is that the expense involved will not be great.

J.T.L.

MEDICINE

Price's Textbook of The Practice of Medicine. Ninth Edition. By Various Authors, Edited by Donald Hunter, M.D., F.R.C.P. Pp. xiv + 1774. £3 3s. 0d. London—New York—Toronto: Oxford University Press. 1956.

Contents: I. Infection and Anti-Bacterial Agents. II. Infectious Diseases. III. Diseases due to Metazoa and Animal Poisons and Venoms. IV. Diseases due to Chemical and Physical Agents. V. Diseases of Metabolism. VI. Deficiency Diseases. VII. Diseases of the Endocrine Glands. VIII. Diseases of the Digestive System. IX. Diseases of the Blood. X. Diseases of the Spleen and Reticulo-endothelial System. XI. Diseases of the Lymphatic System. XII. Diseases of the Cardio-Vascular System. Section XIII. Diseases of the Respiratory System. XIV. Diseases of the Kidney. XV. Diseases of the Joints, Fibrous Tissues and Muscles. XVI. Diseases of the Skeleton. XVII. Diseases of the Skin. XVIII. Diseases of the Nervous System. XIX. Psychological Medicine. Appendix. Index

An old friend appears with an entirely new look. Dr. Frederick W. Price has resigned the editorship after 33 years, during which he produced 8 editions, the last of which was in 1950. Dr. Donald Hunter succeeds him as general editor.

The publishers have done their best to give the book a new look, with a larger page, slightly thicker paper, and a larger and very readable print. This has been done without loss of space, and with reduction in the number of pages from 2,076 in the 8th edition to 1,774 in the 9th edition. The book retains its original thickness but is more convenient and attractive in format. A two-page list of new contributions emphasizes the changes which have resulted from the progress of medicine in 6 years.

In the list of contributors some distinguished names have disappeared through death and resignation, and some new ones have appeared. The contributors are representative mainly of London consultant physicians drawn from many of the medical schools, but there are a few names from outside London. From this list it would be expected that the text would express the best British clinical practice with an emphasis on practice rather than theory. This expectation is fulfilled with a high degree of efficiency, interest and attractiveness, but search for the latest advances in theory in clinical science is sometimes a little unrewarding.

In the discussion on diabetes it is interesting to read that 'our concept of diabetes is approaching a new synthesis', but disappointing to find that this synthesis is 'that diabetes mellitus should be regarded as an inappropriate use of an adaptive protective reaction.' It is interesting to read further on that microaneurysms 'can sometimes be seen as the only change in the retinae of quite young patients who have had diabetes for a number of years', but nowhere is there any discussion of the significance of early and widespread vascular changes in the body in relation to aetiology. It is surprising also to find in a 1956 text-book no reference to the possible significance of carbutamide and its successors in relation to the future treatment of diabetes. On the other hand, the pages on the practical management of diabetes by diet and insulin are very well set out for quick reference.

It would appear that there has been inordinate delay in the completion of the 9th edition, with the result that the text is already out of date in some respects, e.g. carbutamide and its successors in diabetes, mentioned above.

In the section on the thyroid gland recent physiological and biochemical advances are very concisely and accurately summarized. It is surprising, however, to see the multiple nodules of nodular goitre still described as multiple adenomata. In the discussion on malignant change in nodular goitre it is surprising that the difficulties and fallacies of histological interpretation which have caused such differing opinions to be expressed are not discussed. The anxious physician is given no clue as to when he should suspect the possibility of pre-clinical malignancy in a nodular goitre, nor what attitude he should take towards the vexed problem of thyroidectomy in prophylaxis against carcinoma.

The pitfalls of multiple authorship can be illustrated by two examples: sandwiched between two excellent sections on nutritional macrocytic anaemia and kwashiorkor, written by authorities with a world-wide outlook, is a short section on protein deficiency written from the point of view of a British hospital and containing the statement 'protein deficiency rarely results from dietetic insufficiency without an accompanying deficit of calories'. The writer of this section presumably didn't read the following section on kwashiorkor. Secondly, it is somewhat surprising to find erythema nodosum described separately, in somewhat conflicting terms, in the sections on infectious diseases and on dermatology.

In spite of some disappointments the old friend is a good and very welcome friend.

J.F.B.

OBSTETRICS

The Queen Charlotte's Textbook of Obstetrics. Ninth Edition. By Members of the Clinical Staff of The Queen Charlotte's Maternity Hospital, London. Pp. viii + 547. 4 Coloured Plates and 246 Text Figures. 45s. net. London: J. &. A. Churchill Ltd. 1956.

Contents: Foreword. *Section I.—Anatomy and Physiology.* I. Anatomy of the Female Reproductive Organs. II. Physiology of Menstruation, Fertilisation and Embedding of the Ovum. III. Development of the Foetus, Placenta and Membranes. IV. Foetal Physiology. V. Changes in Maternal Anatomy and Physiology during Pregnancy. VI. Anatomy of the Birth Canal and Foetal Skull. *Section II.—Normal Pregnancy.* VII. Signs and Symptoms of Pregnancy. VIII. Ante-natal Care and Management of Pregnancy. *Section III.—Abnormal Pregnancy.* IX. Haemorrhage in Early Pregnancy. X. Vomiting. XI. Albuminuria and Hypertension. XII. Abdominal Pain in Pregnancy. XIII. Abnormalities of Placenta and Membranes. XIV. Abnormalities of Genital Tract during Pregnancy. XV. General Disorders associated with Pregnancy. XVI. Postmaturity. *Section V.—Abnormal Labour.* XIX. Introductory—Delay in Labour. XX. Malpresentations. XXI. The Contracted Pelvis. XXII. Obstructed Labour. XXIII. Abnormal Uterine Action. XXIV. Prolapse of the Cord and Limbs. XXV. Multiple Pregnancy. XXVI. Ante-partum Haemorrhage. XXVII. Post-partum Haemorrhage. XXVIII. Injuries to the Genital Tract. *Section VI.—The Normal Puerperium.* XXIX. The Normal Puerperium. *Section VII.—The Abnormal Puerperium.* XXX. Puerperal Fever. XXXI. Abnormalities of the Puerperium. *Section VIII.—Obstetric Operations* XXXII. Induction of Abortion and Labour. XXXIII. Forceps and Version. XXXIV. Caesarean Section. XXXV. Destructive Operations. *Section IX.—The Baby.* XXXVI. The Healthy Baby. XXXVII. The Premature Baby. XXXVIII. The Sick Baby. *Section X.—Miscellaneous.* XXXIX. The Rhesus Factor. XL. Psychiatry and Reproduction. XLI. Oxytocic Drugs in Midwifery. XLII. Anaesthesia and Analgesia in Obstetrics. XLIII. Radiology

in Obstetrics. XLIV. Maternal Mortality. XLV. Foetal and Neonatal Mortality. XLVI. Post-Natal Care. Index.

This orthodox British text-book of obstetrics has now reached its 9th edition since it was first published in 1927—ample evidence of its popularity. It is written by 15 members of the staff of Queen Charlotte's Hospital. Particular care has been taken to retain the practical quality of the book and all the methods described are those that are now taught at the hospital. The editor (S. G. Clayton) has accepted the responsibility for the selection of the material in those instances in which the opinions of the staff differed.

The book covers the field of obstetrics in a comprehensive yet compact manner and, although all will agree with most of the opinions expressed, a few comments are necessary. In the treatment of atonic postpartum haemorrhage no mention is made of the value of the 'pitocin drip'—a procedure of great value. Eclampsia is treated in the orthodox fashion, the authors stressing that the prevention of further convulsions should be the all-important aim of therapy. They are not impressed with the value of hypotensive drugs in the treatment of this condition, and they emphasize the value of good nursing care. In a practical way they list the conditions under which the patient can be disturbed. No mention is made of pentothal anaesthesia in the primary hospital treatment of the condition, and for the induction of labour artificial rupture of the membranes is recommended, but not until the fits have been controlled for at least 48 hours. Recently, the immediate induction of labour, under pentothal anaesthesia, by rupture of the membranes is being tried in this country. The results so far have been satisfactory. One is still puzzled why the term 'morphia' is used in text-books instead of the correct term 'morphine'. The authors still recommend Credé's expression for retained placenta and condemn cord-pulling. The very useful Brandt-Andrews manoeuvre is not mentioned. The recommendation that a Wertheim operation, preceded by a Caesarean section, be done for the treatment of cervical carcinoma in late pregnancy, will not find favour with all. The technical difficulties at that stage of pregnancy are sometimes considerable. The difficulty of diagnosis of postmaturity is emphasized, yet the majority of the authors recommend induction of labour by articial rupture of the membranes at the 42nd week of gestation.

One feels that altogether this is a good book in the true tradition of British obstetrics—what else could be expected when it comes from Queen Charlotte's?

W.H.M.

CORRESPONDENCE : BRIEWERUBRIEK

DISEASE INCIDENCE IN GENERAL PRACTICE

To the Editor: I greatly enjoyed the excellent and most interesting paper under this title by my good friend Dr. P. J. van Biljon[1] appearing on page 397 of the *Journal* of 27 April 1957.

I would point out to him, however, that whereas his incidences of 26·2% and 26·4% respectively for medical and surgical consultations apply to men, women and children seen, and the latter percentage is, as he admits, weighted by industrial injuries, his 8·7% gynaecological and obstetrical cases apply to women only and comprise 20·4% of all the conditions for which his female patients consulted him.

It would be interesting to compare this corrected incidence of gynaecological and obstetrical consultations with those for medical and surgical conditions and the 'minor subjects' in women only. With the greater preponderance, as demonstrated, of gynaecological consultations, the medical and surgical incidences would be correspondingly less and probably also around 20%. A woman, at least then, if not her husband or child, should be entitled to expect that as much time should have been spent in training her doctor in those diseases peculiar to her alone, and in the art of bringing healthy children into the world for both her husband and herself to enjoy.

I hope that in the years to come Dr. Van Biljon's obstetrical and gynaecological side of his practice will so flourish that even without these necessary corrections there will be no danger of his relegating our speciality to the 'minor subjects'.

J. P. Roux

Department of Obstetrics and Gynaecology
Karl Bremer Hospital
Bellville, Cape
29 April 1957

1. van Biljon, P. J. (1957): S. Afr. Med. J., **31**, 397.

PENSION SCHEME *VS.* ENDOWMENT ASSURANCE

To the Editor: Last year a sub-committee appointed by the Cape Western Branch of the Association investigated the possibility of a pension scheme for the profession, to enable doctors who are self-employed to set aside a percentage of their annual income for pension purposes. This percentage of annual income set aside could be free of Income Tax only if the scheme gained Government recognition. This investigation was cut short by a circular from the Association.

The circular to members announced that Federal Council was not prepared to sponsor any pension scheme but that, on application to the Association, members could obtain particulars of an attractive endowment assurance policy, which would serve as an *excellent* pension scheme.

This untimely circular was indeed doubtful advice. Firstly, Federal Council should try with determination to obtain a recognized pension scheme which has no connection with insurance companies. Secondly, the Insurance Agency of the Association is paid a commission for policies it sells to the profession, and so it should not give doubtful advice, from which the Association shall benefit.

However, the Professional Provident Society has now determined to press for such a pension scheme, and anyone interested is well advised to correspond with this non-profit society, which is run by doctors themselves.

For years the Atlas Assurance Company has enjoyed the blessing of the South African Medical Association and in a very recent issue of the *Journal* this company is again recommended. Yet there is another very reputable assurance company which gives all the cover that the Atlas does, and for a much smaller premium.

I put it to the South African Medical Association that should it wish to maintain the confidence of its members, it must refrain from issuing advice of doubtful value, especially where it is done by a special circular as if to carry real weight. One does not doubt the integrity of those concerned. The benefits reaped by the Association from business arrangements are to be encouraged, but, please, not to the detriment of innocent members.

John H. Bam

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St. George's Street
Cape Town

COORDINATING THE NATIONAL WORK OF REHABILITATION

To the Editor: On page 233 of the *Journal* of 9 March 1957 there is an article[1] by your Parliamentary Correspondent under this title. In this article appears the following paragraph:

'This draft Bill will be submitted by me to the Government as soon as a gentleman's agreement has been reached with the mining industry that mineworkers who had been injured in accidents while working on a mine would be re-employed by the industry.'

As far as I am aware the official report of the debate in respect of this paragraph in particular reads:

'A gentleman's agreement has been entered into in the mining industry by which employees who are in the service of the mines will once again be able to be employed by those mines should some accident or other befall them.'

C. J. Goedvolk
Medical Superintendent

The Chamber of Mines Hospital
P.O. Box 774
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23 April 1957

1. Report (1957): S. Afr. Med. J., **31**, 233.